JAN 26 1944

The Journal of Bone and Joint Surgery

The Official Publication of
The American Orthopaedic Association
The British Orthopaedic Association
The American Academy of Orthopaedic Surgeons
Owned and Published by The American Orthopaedic Association

Medical Library

January 1944

8 THE FENWAY, BOSTON, MASSACHUSETTS, U. S. A.

Vol. XXVI, No. 1 *Published Quarterly* Old Series: Vol. XLII, No. 1

The Journal of Bone and Joint Surgery

The Official Publication of
THE AMERICAN ORTHOPAEDIC ASSOCIATION
THE BRITISH ORTHOPAEDIC ASSOCIATION
THE AMERICAN ACADEMY OF ORTHOPAEDIC SURGEONS
Owned and published by The American Orthopaedic Association

VOLUME XXVI
OLD SERIES - VOLUME XLII
1944

8 THE FENWAY, BOSTON 15, MASSACHUSETTS, U. S. A.

Medical
Navy

The Journal of Bone and Joint Surgery*

The Official Publication of
The American Orthopaedic Association
The British Orthopaedic Association
The American Academy of Orthopaedic Surgeons
Owned and published by The American Orthopaedic Association
*Title registered in United States Patent Office

VOL. XXVI, No. 1 *January, 1944* **Old Series VOL. XLII, No. 1**

Editor, pro tempore, CHARLES F. PAINTER, M.D. *Assistant Editor,* FLORENCE L. DALAND

BOARD OF ASSOCIATE EDITORS

G. E. HAGGART, M.D.
LEO MAYER, M.D.
FRANK R. OBER, M.D.
CHARLES F. PAINTER, M.D.
WILLIAM A. ROGERS, M.D.
A. R. SHANDS, JR., M.D.

MR. NORMAN L. CAPENER, F.R.C.S., *British Editorial Secretary.*
The Princess Elizabeth Orthopaedic Hospital, Exeter, England

EDITORIAL COMMITTEE

FRANK D. DICKSON, M.D., *Treasurer,* 1400 Professional Building, Kansas City, Missouri
CHARLES W. PEABODY, M.D., *Secretary,* 3001 West Grand Boulevard, Detroit, Michigan
JOHN ALBERT KEY, M.D., *President-Elect,* Washington University School of Medicine, St. Louis, Missouri
H. J. SEDDON, F.R.C.S., *Secretary,* Wingfield-Morris Orthopaedic Hospital, Headington, Oxford, England

Published quarterly: January, April, July, and October
8 THE FENWAY, BOSTON, MASSACHUSETTS

¶ ORIGINAL ARTICLES. — Papers for publication will be accepted only for exclusive publication in this Journal. The American Orthopaedic Association does not officially endorse the opinions presented in the different papers published in this Journal.

¶ COPYRIGHT. — Material appearing in The Journal is covered by copyright, but, as a general rule, no objection will be made to the reproduction in reputable medical journals of anything in these pages, provided that permission is obtained from The Journal and that proper credit is given.

¶ ADVERTISEMENTS. — Advertising rates will be furnished on application.

¶ CHANGE OF ADDRESS. — In sending change of address, both the old and new addresses should be given. ¶ THE SUBSCRIPTION PRICE of this Journal is $5.00 per year, payable in advance, for the United States and such other countries as, under the revised postal regulations, are subject to domestic rates; $5.25 for Canada; the equivalent of $5.75 for all other foreign countries included in the postal union. Single copies of current issue $1.50. ¶ Checks, drafts, and post-office money orders should be made payable to The Journal of Bone and Joint Surgery.

Address all Communications to the Editor
THE JOURNAL OF BONE AND JOINT SURGERY
8 The Fenway, Boston 15, Massachusetts, U. S. A.

TABLE OF CONTENTS

PAGE

OBSERVATIONS ON THE FUNCTION OF THE SHOULDER JOINT.
By *Verne T. Inman, M.D., J. B. deC. M. Saunders, F.R.C.S.,* and *LeRoy C. Abbott, M.D.,* San Francisco, California 1

LESIONS OF THE MUSCULOTENDINOUS CUFF OF THE SHOULDER. I. THE EXPOSURE AND TREATMENT OF TEARS WITH RETRACTION.
By *Harrison L. McLaughlin, M.D.,* New York, N. Y. 31

BLADE-PLATE INTERNAL FIXATION FOR INTERTROCHANTERIC FRACTURES.
By *Austin T. Moore, M.D.,* Columbia, South Carolina 52

SULFONAMIDES IN THE TREATMENT OF CHRONIC OSTEOMYELITIS.
By *J. Albert Key, M.D.,* St. Louis, Missouri 63

CAPSULECTOMY FOR THE RELIEF OF FLEXION CONTRACTURES OF THE ELBOW FOLLOWING FRACTURE.
By *Philip D. Wilson, M.D.,* New York, N. Y. 71

THE KENNY CONCEPTS AND TREATMENT OF INFANTILE PARALYSIS.
By *A. Bruce Gill, M.D.,* Philadelphia, Pennsylvania 87

EXPERIENCES WITH THE KENNY TREATMENT FOR ACUTE POLIOMYELITIS IN THE EPIDEMIC OF 1942, MONMOUTH AND OCEAN COUNTIES, NEW JERSEY.
By *Nicholas S. Ransohoff, M.D.,* New York, N. Y. 99

ANALYSIS OF NEUROMUSCULAR DISORDERS IN POLIOMYELITIS.
By *Joseph Moldaver, M.D.,* New York, N. Y. 103

METAL-BLOCK REPLACEMENT OF BONE DEFICIENCY. A PRELIMINARY REPORT ON AN OPERATIVE CORRECTION FOR GENU RECURVATUM.
By *Ross Sutherland, M.D.,* Los Angeles, California, and *M. John Rowe, Jr., M.D.,* Long Beach, California 118

CHIP FUSION OF THE LOW BACK FOLLOWING EXPLORATION OF THE SPINAL CANAL.
By *Henry Briggs, M.D.,* and *Paul R. Milligan, M.D.,* East Orange, New Jersey 125

CARPECTOMY FOR INTRACTABLE FLEXION DEFORMITIES OF THE WRIST.
By *J. Warren White, M.D.,* and *Sam G. Stubbins, M.D.,* Greenville, South Carolina 131

COMPRESSION FRACTURES OF THE VERTEBRAL BODIES AND OTHER CHANGES MISTAKEN FOR THEM.
By *John D. Ellis, M.D.,* Chicago, Illinois 139

MARCH FRACTURE. REPORT OF A CASE INVOLVING BOTH FIBULAE.
By *Lieutenant Colonel A. Scott Hamilton* and *Major Howard E. Finklestein,* Camp Claiborne, Louisiana 146

(Continued on page 19 following Current Literature)

VOL. XXVI, NO. 1 JANUARY, 1944 Old Series Vol. XLII, No. 1

The Journal of Bone and Joint Surgery

OBSERVATIONS ON THE FUNCTION OF THE SHOULDER JOINT * †

BY VERNE T. INMAN, M.D., PH.D., J. B. DEC. M. SAUNDERS, F.R.C.S., AND LEROY C. ABBOTT, M.D., F.A.C.S., SAN FRANCISCO, CALIFORNIA

From the Divisions of Orthopaedic Surgery and Anatomy, University of California Medical School, San Francisco

The manifest complexity of the mechanism of the shoulder joint is evident to anyone who has watched the progression of what Codman has so aptly described as "scapulohumeral rhythm". This rhythm is participated in by the whole complex of joints constituting the shoulder girdle. Therefore, the term shoulder joint is somewhat misleading, unless we clearly bear in mind that this expression includes no less than four different joints: the sternoclavicular, the acromioclavicular, the scapulothoracic, and the glenohumeral; and that motion at the shoulder is the sum of movement contributed by synchronous participation of all these joint units. In the development of rational procedures for the correction and reconstruction of disabilities affecting the shoulder mechanism, it is necessary to break down this complex into its various components in order that we may uncover some of the fundamental principles underlying their action.

Many such analyses have been carried out in the past, but none, so far as we are aware, have attempted to solve or derive a comprehensive picture of the whole. Much of the early work is very contradictory, and nearly all is incomplete, due to lack of an adequate experimental method. For this reason many misconceptions exist, owing to the too ready facility with which investigators have evolved conceptions based on *a priori* reasoning from the inert cadaver. In studying functional mechanisms, there is only one touchstone to which we can appeal, and that is the living body. It is this appeal which enlivens the observations derived from the cadaver to which nonetheless we are compelled to turn on occasion for certain basic information.

It is with such dynamic considerations uppermost, acting as the unifying theme, that we have attacked the problem anew, so as to obtain an unbiased and more vital point of view. Logically and chronologically, we have, therefore, examined the shoulder mechanism from several aspects,—namely the comparative anatomical, the roentgenographic analysis of motion, the theoretical force requirements, and the action current potential derived from the living muscle in motion, and from the data so obtained have attempted a resynthesis of the whole. Our studies have been rendered possible, thanks to the generosity of The National Foundation for Infantile Paralysis to whose support we acknowledge our deep indebtedness.

* Read at the Annual Meeting of The American Orthopaedic Association, Cleveland, Ohio, June 8, 1943.
† This work was done under a grant from The National Foundation for Infantile Paralysis, Inc., New York, N. Y.

In presenting this brief summary of our findings and conclusions, the chronological order in which the mechanism was investigated will be followed, as being the most logical and coherent approach.

COMPARATIVE ANATOMY

The Scapula

On examining the skeletal elements of those forms which have freed the forelimb for prehensile purposes, certain very striking trends are discernible. The scapula in the living form is suspended, as it were, in space by the muscles acting upon it, and it is, therefore, not surprising that this bone should reflect more clearly than any other the changes which have been brought about by more specialized functional demands. The most striking and obvious modifications are those which have occurred in scapular shape. Those alterations can best be expressed by an index known as the scapular index, indicating the relationship of length to breadth of the bone (Fig. 1). This index is extremely high in the pronograde, where the scapula is long and narrow, but due to increasing breadth, it progressively falls as we approach the orthograde, such as man, in which the forelimb has been completely freed. The same tendency can be seen in forms, other than the primates, such as the rat and marsupial, in which similar but partial freeing of the forelimb has also occurred. It will be noticed that the changes in shape are almost exclusively confined to that portion of the scapula which lies below its spine. These are demonstrated by the infraspinous index which, therefore, progressively rises. The extension of the infraspinous fossa necessarily alters the relationship of the axillary border of the scapula to the glenoid and, in consequence, the angle of action of its attached muscles, thereby establishing a feature of great importance in the shoulder mechanism. The supraspinous portion of the scapula is very conservative, and is altered but little in the proportion of the scapula which it makes up. Other changes of particular significance in the primate series are those associated with morphological alterations in the size and extent of the acromion process (Fig. 2).

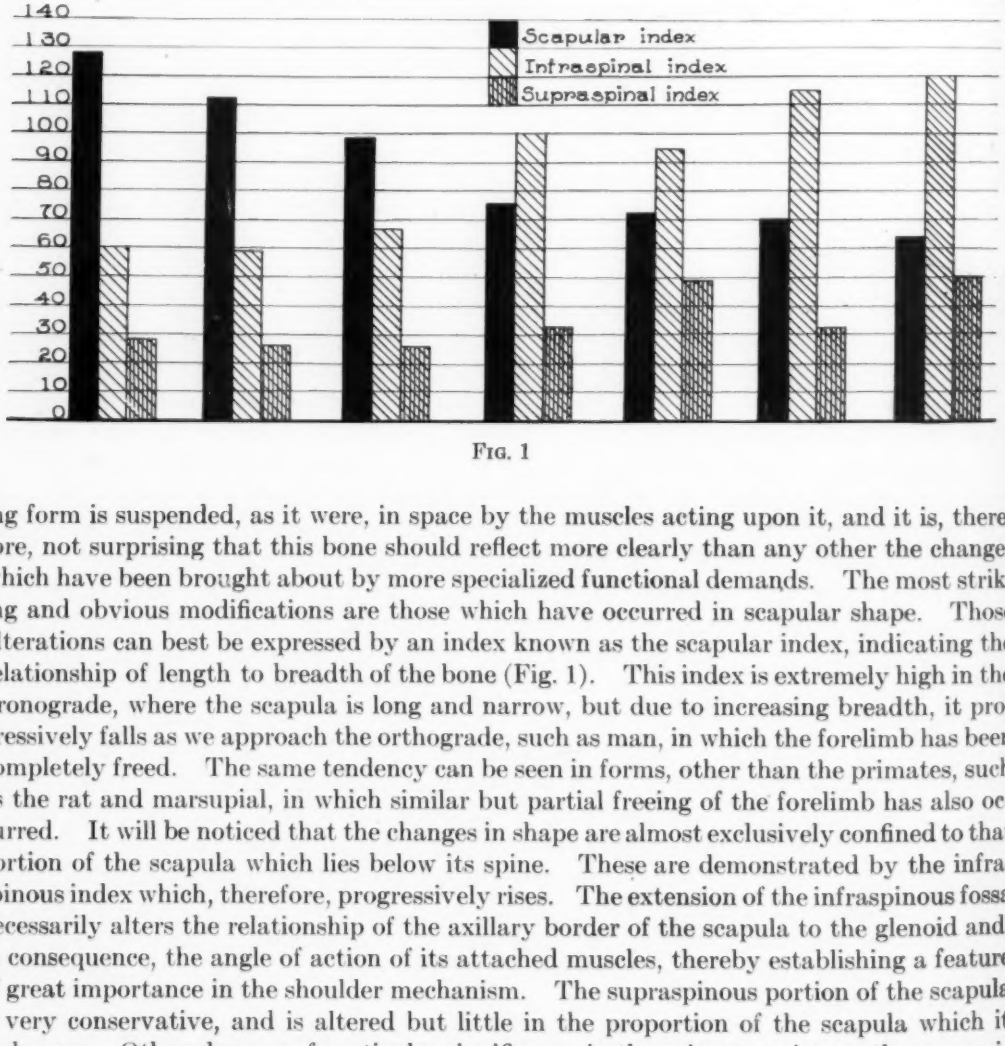


FIG. 1

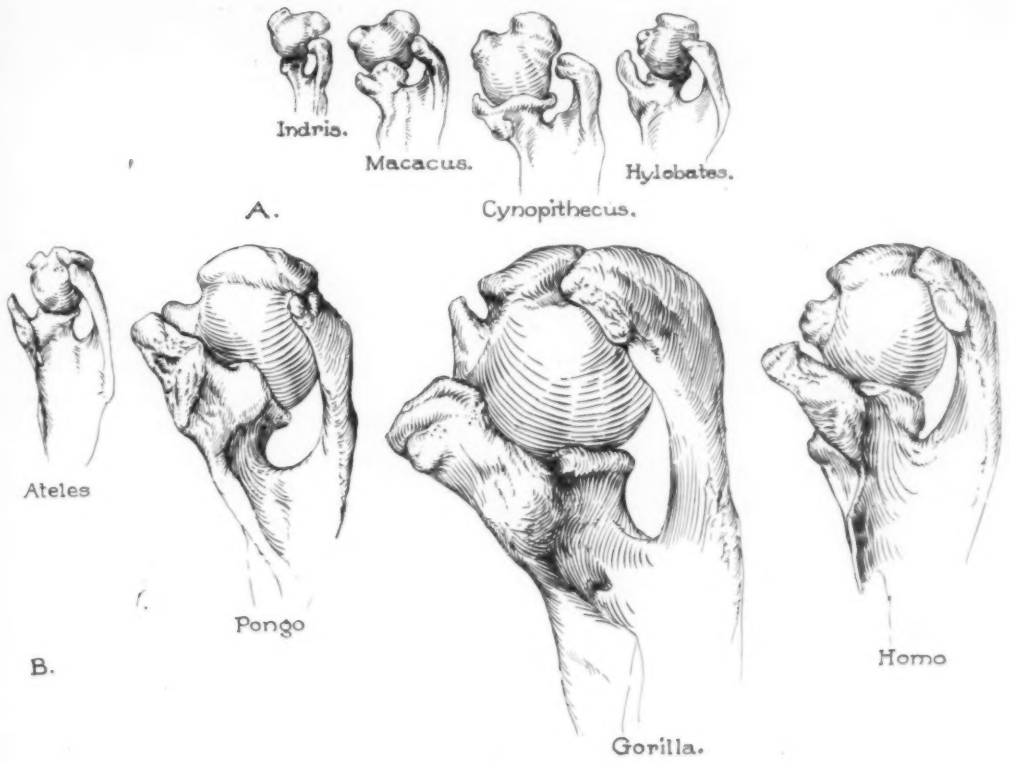


FIG. 2

This process in more primitive forms is short and delicate, projecting for but a short distance over the head of the humerus. As we approach the anthropoids, it increases in mass and size, and projects over the head of the humerus so as to conceal it almost entirely.

In summary, those mammals which have freed the forelimb exhibit alterations in the scapula, all of which point in the same general direction. The long, narrow scapula of the pronograde form has become progressively broader, due almost exclusively to the relative extension and increase in size of the infraspinous fossa. On the other hand, the supraspinous fossa has remained unaltered. The extension of the infraspinous fossa is undoubtedly related to the change in functional requirements of the attached muscles and, in addition, emphasizes, as will be shown later, the extraordinary significance of the infraspinal musculature in the attainment of a shoulder joint with the great range of motion of that found in man. Likewise, the progressive increase in size, mass, and extension of the acromion process indicates the more dominant position occupied by the deltoid.

The Humerus

Like the scapula, the humerus exhibits certain fundamental alterations in morphology. One of the most obvious is the progressive distal migration of the point of insertion of the deltoid muscle, which, with the increasing size of the acromion process of the scapula, extends the functional advantage of the muscle and reflects the

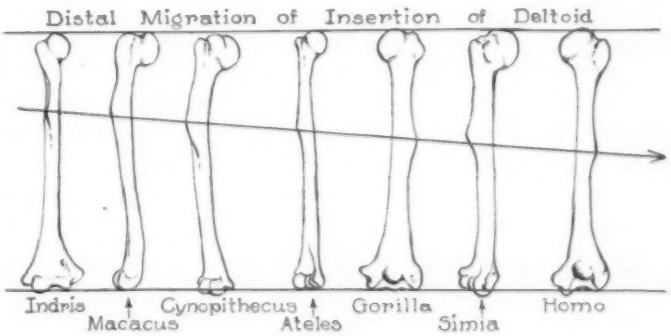


FIG. 3

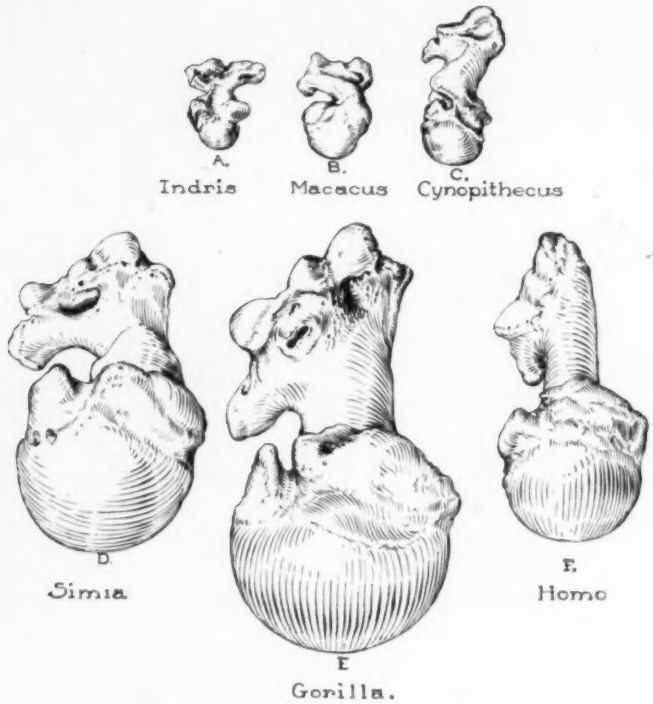


FIG. 4

increasing importance which the deltoid will progressively assume in the mechanism of a free limb (Fig. 3).

A further characteristic change in the humerus is the development of torsion in its shaft (Fig. 4). In quadrupeds the articular surface of the head of the humerus is directed dorsally, and an imaginary line, connecting the epicondyles at the elbow, lies in the coronal plane of the body. These two axes, therefore, make an angle of approximately 90 degrees. As the thoracic cage flattens with the erect posture, the scapula is rotated dorsally. With the necessity of maintaining the elbow and its movements of flexion in the parasagittal plane, the head of the humerus has, perforce, to follow the scapula in its displacement on the chest wall. The humerus, therefore, undergoes torsion; and the axes already mentioned now come, in man, to make an angle of approximately 164 degrees.

The torsion of the humerus has, in turn, affected the position and size of the humeral tubercles. In primitive forms, the bicipital groove lies approximately midway between the two tubercles, which are of almost equal size. The effect of the torsion has been to displace the bicipital groove medially, so that it encroaches upon, and reduces the size of the lesser tubercle.

The distal migration of the insertion of the deltoid and the torsion of the humeral shaft, together with the change in position of the bicipital groove, have profoundly affected the musculature of the shoulder, particularly that of the long-headed biceps. The significance of these alterations will become apparent as the functional relationship of the muscles is studied.

THE MUSCLES

Associated with modifications of the skeletal parts, there have been striking alterations in the muscles, in respect to both their size and points of attachment, which suggest that the functional demands of the extremity, expressing themselves through the action of the muscles, have caused the skeletal changes.

The changes in the musculature are most evident when the relative mass of an individual muscle is compared to the total mass of the group. Of muscles acting in the mechanism of the shoulder, there are three topographical groups: (1) those passing from the scapula to the humerus, the scapulohumeral muscles; (2) those passing from the trunk to the humerus, the axiohumeral group; and (3) those passing from the trunk to the scapula, the axioscapular group.

The Scapulohumeral Group

The first category comprises the supraspinatus, infraspinatus, teres minor, subscapularis, deltoid, and teres major.

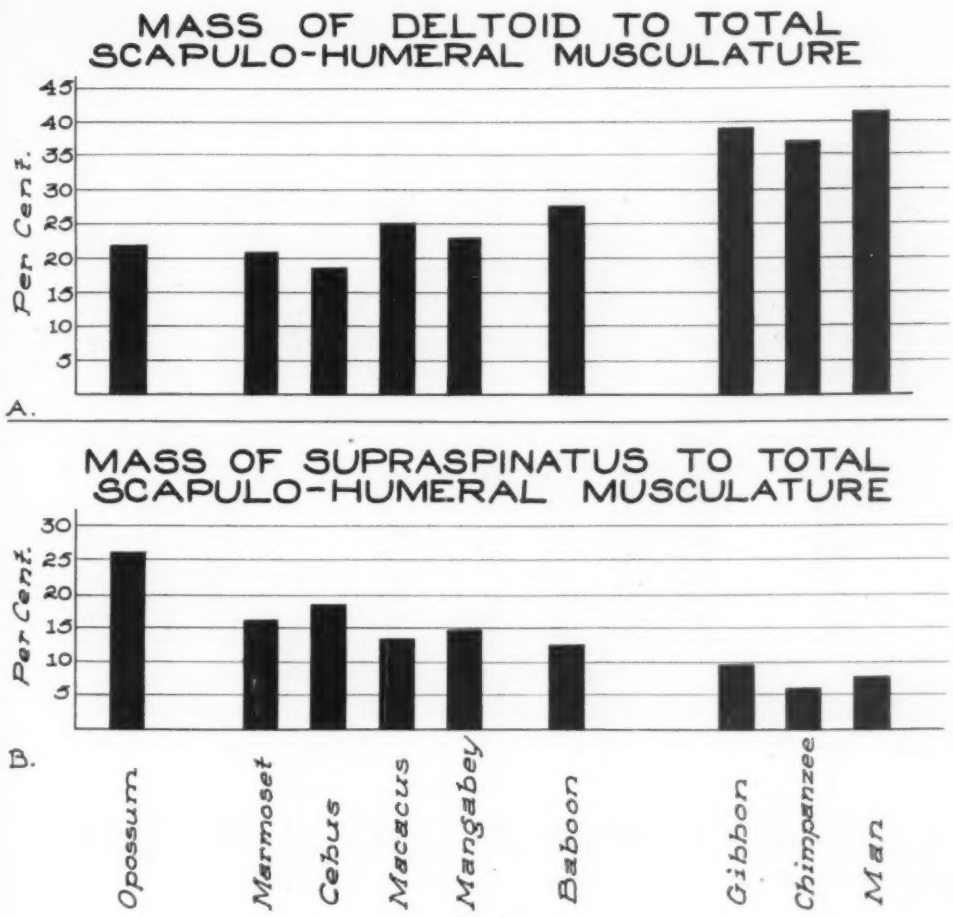


FIG. 5

Of these, as we pass from the lower primates to the anthropoids, we find that the supraspinatus, while remaining relatively static in morphology, has progressively decreased in relative mass (Fig. 5*B*).

On the other hand, the deltoid muscle in man has increased very significantly in proportional mass (Fig. 5*A*). In a generalized mammalian form, such as the opossum, it makes up approximately 20 per cent. of the total scapulohumeral musculature. In man, however, its proportional representation has more than doubled, and the muscle now makes up approximately 41 per cent. of this complex. A similar increase in the relative size of the muscle is exhibited by other series, as in the rat family, where a like freeing of the forelimb has occurred.

The changes in scapular shape have profoundly influenced the morphology of the deltoid muscle in another direction. That portion of the muscle which is attached to the region of the inferior angle of the scapula in primitive forms, has followed its distal extension, and has thus become separated from the general mass. In this way a portion of the deltoid muscle is established as a morphological separate element,—namely, the teres minor. Comparative anatomical investigations clearly reveal that the teres minor is a morphological component of the deltoid muscle.

The power of the deltoid muscle has been increased in two ways: first, by an increase in both absolute and relative size; and second, by the increased leverage achieved by the development of the acromion and by the distal migration of the deltoid insertion.

As we have already indicated, the teres minor is morphologically a portion of the deltoid. This muscle is, therefore, absent in the primitive mammalian. As the infraspinous

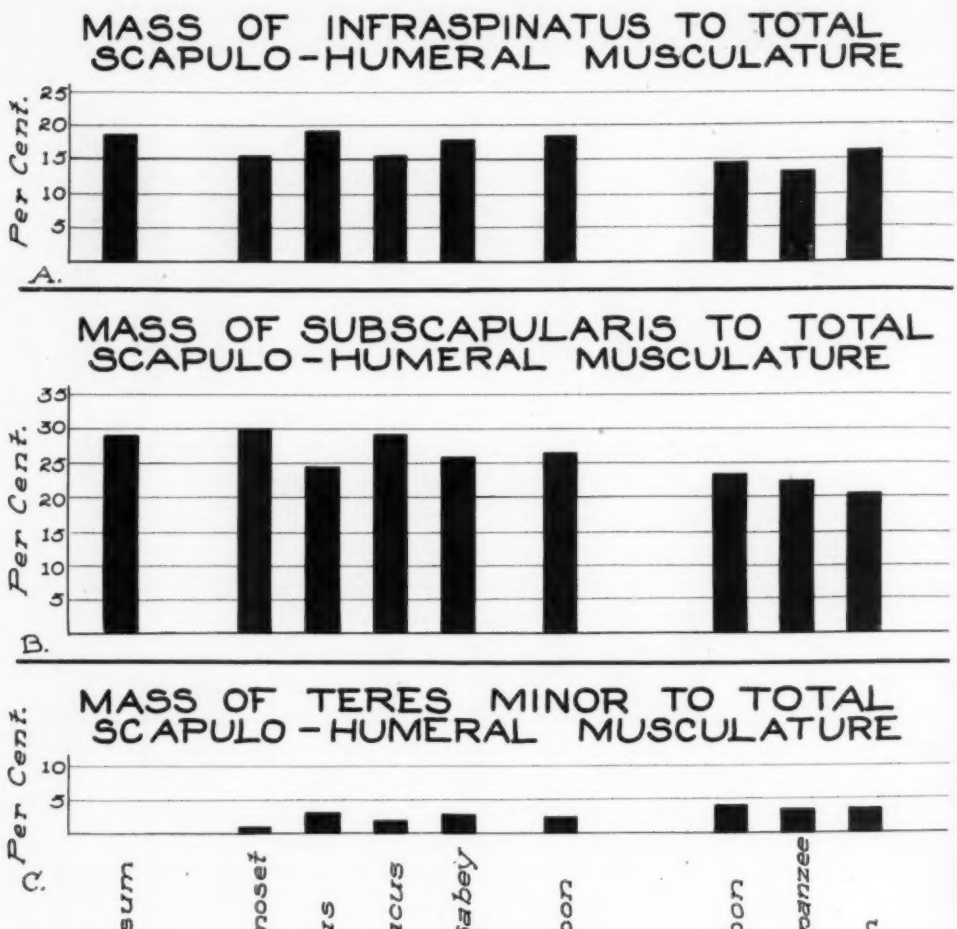


FIG. 6

fossa of the scapula increases, and this element of the muscle separates from the deltoid, it also begins to show progressive increase in mass (Fig. 6*C*). Absent from the scapulohumeral musculature of the opossum, it, however, makes up nearly 5 per cent. in man. In addition, the skeletal changes have altered the position of the teres minor, so that it comes to act upon the humerus in a downward direction, having been displaced through almost a right angle. This, as will be shown later, is necessary to fulfill a fundamental requirement in the freeing of the shoulder.

In primitive forms, the subscapularis is the largest muscle of the scapulohumeral group. For example, in opossums, it is larger than the deltoid, making up 28 per cent. of the shoulder musculature; the deltoid only 22 per cent. As we pass through the primate group to man, the subscapularis decreases relatively but slightly (Fig. 6*B*). It undergoes, however, some morphological alteration by increasing the number of its fasciculi as its origin expands with the increasing extension of the scapula. As a result, the lowermost fasciculi, like those of the teres minor, act upon the humerus in a downward direction.

The infraspinatus behaves in much the same manner as the subscapularis by extending its area of attachment; for like, the subscapularis, it is influenced by the elongation of the scapula (Fig. 6*A*). We find, therefore, that the teres minor, the subscapularis, and the infraspinatus form a functional group, and they, by morphological extension of the scapula,

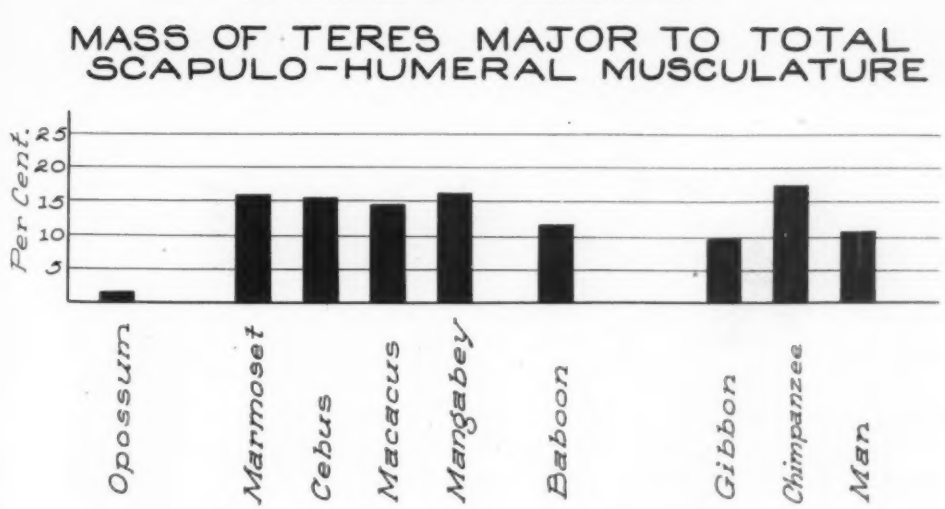


FIG. 7

can now exercise their function as depressors of the head of the humerus as well as rotators.

There is no definite trend to be noted in the teres major, except to observe that this muscle is large in climbing forms (Fig. 7).

The Axioscapular Group

The axioscapular muscles are an important and distinctive group in the functional mechanism of the shoulder, and consist predominantly of the trapezius, the rhomboids, the serratus anterior, and the levator scapulae.

Throughout the primate series, the trapezius has altered surprisingly little, both in proportionate mass and in general morphological arrangement. The only significant change has been the concentration of the muscle elements along its upper and lower borders, which is associated with a developing deficiency in the intermediate part of the muscle lying opposite the spine of the scapula.

The serratus anterior and the levator scapulae, on the other hand, constitute a single sheet of muscle in primitive forms, passing from the transverse processes of the cervical vertebrae and the upper eight or ten ribs to the vertebral border of the scapula (Fig. 8*A* and *B*). Progressive loss of the intermediate portion divides the sheet and leads to its separation into the definitive levator scapulae and serratus anterior (Fig. 8*C*). The serratus, once defined, in turn exhibits morphological changes similar to those of the trapezius, for, with progressive diminution in the mass and significance of its intermediate digitations, there is a trend toward concentration of the muscle insertion at the superomedial and inferior angles of the scapula, thereby establishing an upper and lower functional component (Fig. 8*D*). It will be shown that these morphological alterations in the serratus and levator scapulae sheet are definitely related to functional requirements of the shoulder mechanism, and that the action of a single muscle, such as serratus, differs in its individual parts.

There is little significant change in the rhomboids.

The Axiohumeral Group

The axiohumeral group of muscles connecting the humerus to the trunk, comprises the pectoralis major and minor and the latissimus dorsi.

The pectoral muscles were originally derived from a single mass, which underwent separation into superficial and deep layers. The superficial layer is the pectoralis major whose origin has partially migrated in a cephalic direction, thus establishing the clavicular head of the muscle. In addition, concentration of an originally more diffuse insertion provides the characteristic bilaminar tendon of man.

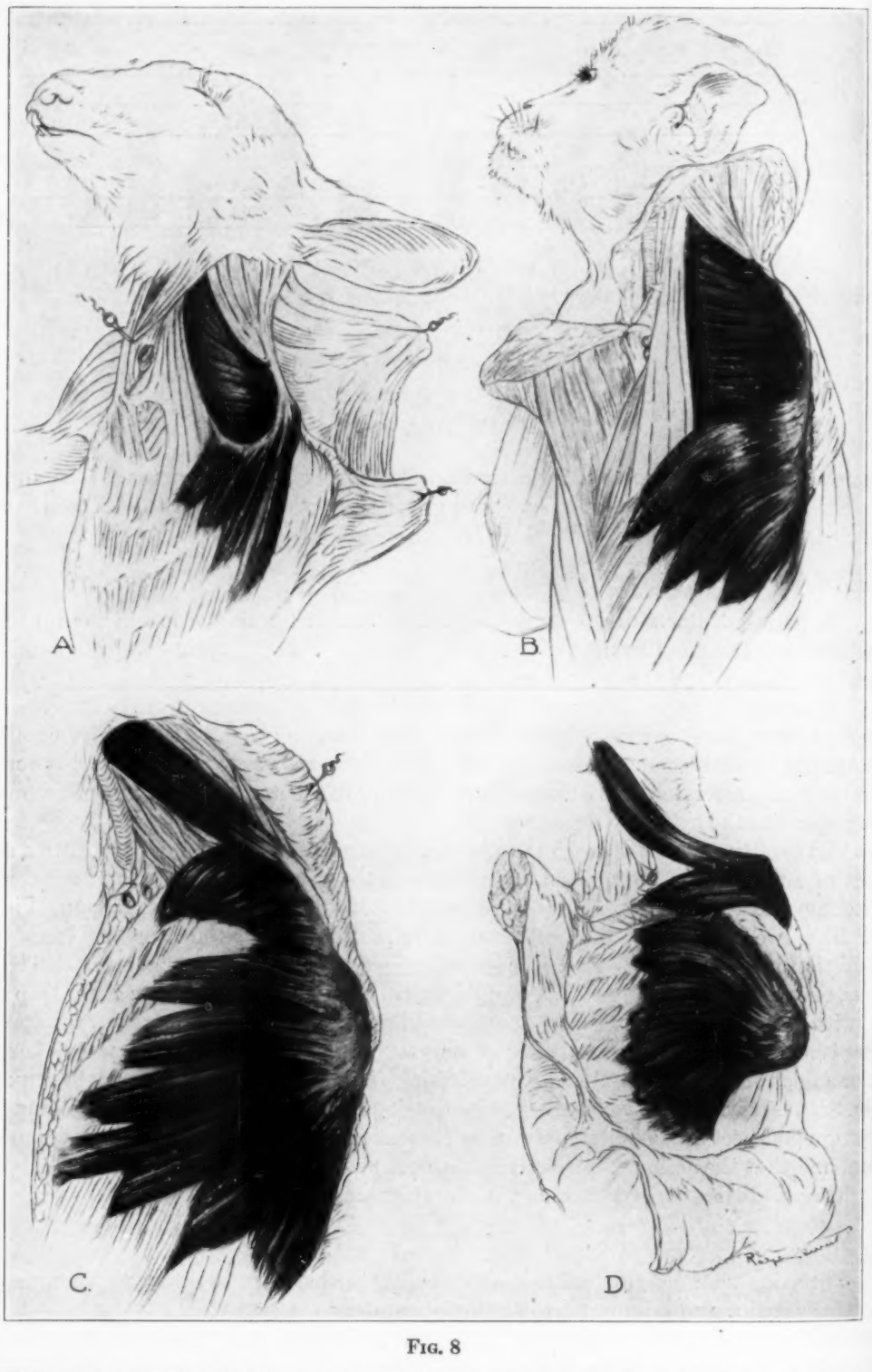


FIG. 8

The segregation of the levator scapulae and the serratus anterior from a primitive muscle sheet, arising from the ribs and the transverse processes of the cervical vertebrae.
A: Wallaby. *B:* Macacus. *C:* Man. *D:* Gibbon.

The deep layer gives rise to the pectoralis minor, which in most forms is attached to the humerus, but this attachment has undergone progressive transference to the coracoid process of the scapula, so that, in chimpanzee and man, this muscle topographically belongs to the axioscapular group. An interesting development of a new structure is associated with the transference of the insertion of this muscle. As the freedom of the limb increases, the accompanying enlargement and extension of the acromial arch results in the development of the subdeltoid bursa. This bursa, familiar to every orthopaedic surgeon, appears for the first time in the chimpanzee.

The remaining muscle of this group, the latissimus dorsi, is morphologically very conservative and, like the teres major, is most massive and extensive in climbing forms.

Muscles of the Upper Arm

The biceps and triceps belong to a special category in that they extend from the scapula to the bones of the forearm. In primitive mammals, the biceps has but a single head of origin. In certain specialized forms, such as the horse, in which the deltoid is absent, a large and single-headed biceps is utilized in the elevation of the extremity in conjunction with a massive supraspinatus. It is in the primates that we find two heads of origin make their appearance. The long head is, however, displaced medially as a result of the torsion occurring in the shaft of the humerus, but if, in man, the arm is fully rotated externally, thus restoring the primitive relative position of the tendon, the muscle can once again be utilized as an abductor of the shoulder,—a phenomenon often witnessed in infantile paralysis when all other abductors of the scapulohumeral joint have been lacking.

The triceps is retained in its primitive relationship and morphology, but the size of its long head has been progressively reduced.

MOTION AT THE SHOULDER JOINT

An essential preliminary to the analysis of the mechanism of the shoulder is an understanding of the sequence of motion which occurs at its component joints. The shoulder joint complex is composed of four independent articulations,—the sternoclavicular, acromioclavicular, scapulothoracic, and glenohumeral joints. While each of these is an independent entity, capable of independent motion, all contribute their share to the total in the normal functional mechanism of the extremity. Furthermore, the participation of each of these joints in the entire movement is simultaneous, and not successive.

We have employed for the elucidation of these movements both roentgenography and the direct insertion of pins into the bones of the living subject.

Elevation of the extremity, both in flexion and in abduction, at the glenohumeral articulation is simultaneously accompanied by scapulothoracic movement, an arrangement which critically enhances the power of the attendant muscles. In the first 30 to 60 degrees of elevation, the scapula seeks, in relationship to the humerus, a precise position of stability which it may obtain in one of several ways. Either the scapula remains fixed, motion occurring at the glenohumeral joint until the stable position is reached, or the scapula moves laterally or medially on the chest wall, or in rare instances oscillates until stabilization is attained. Hence the early phase of motion is highly irregular, and is characteristic for each individual. It would seem to depend upon the habitual position which the scapula occupies in the subject when at rest. This phase of motion is related to the setting action of the muscles, and we have, therefore, termed it "the setting phase".

Once 30 degrees of abduction, or 60 degrees of forward flexion has been reached, the relationship of scapular to humeral motion remains remarkably constant (Figs. 9 and 10). Thereafter a ratio of two of humeral to one of scapular motion obtains; and thus between 30 and 170 degrees of elevation, for every 15 degrees of motion, 10 degrees occurs at the glenohumeral joint, and 5 degrees by rotation of the scapula on the thorax.

It should be clearly recognized that the orthodox teaching on these motions is entirely

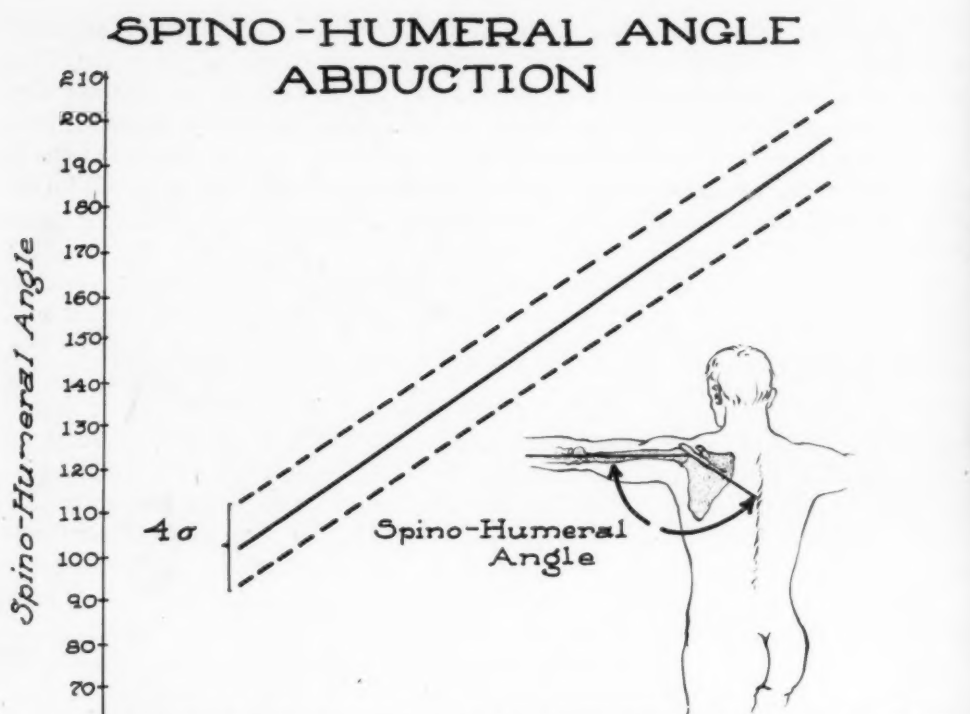

SPINO-HUMERAL ANGLE
ABDUCTION
Spino-Humeral Angle
210
200
190
180
170
160
150
140
130
120
110
100
90
80
70
4σ
Spino-Humeral
Angle
10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180 190
Degree of Elevation of Arm

FIG. 9

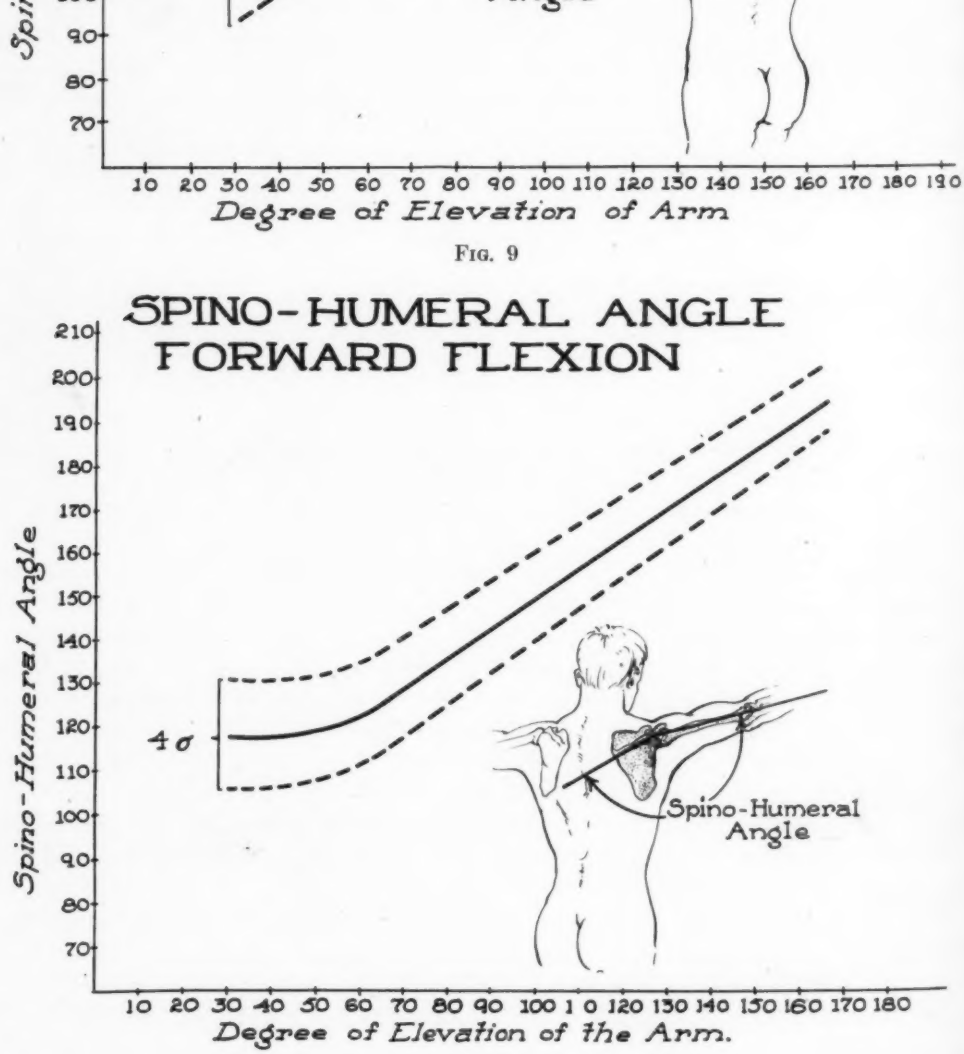

SPINO-HUMERAL ANGLE
FORWARD FLEXION
Spino-Humeral Angle
210
200
190
180
170
160
150
140
130
120
110
100
90
80
70
4σ
Spino-Humeral
Angle
10 20 30 40 50 60 70 80 90 100 1 0 120 130 140 150 160 170 180
Degree of Elevation of the Arm.

FIG. 10

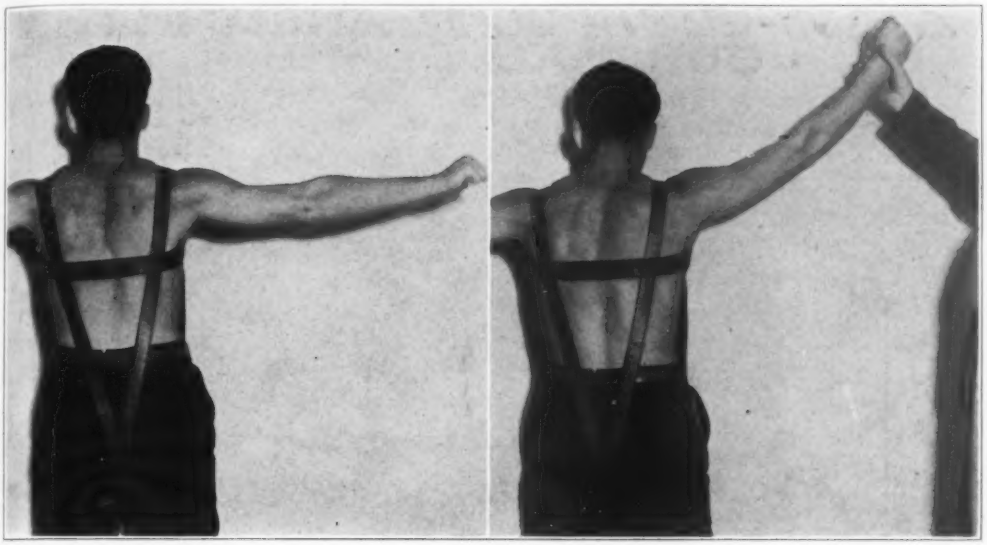


FIG. 11

incorrect. The standard textbooks state that glenohumeral motion occurs up to the right angle and that thereafter further elevation is brought about by rotation of the scapula. Roentgenography and examination of the living prove beyond a doubt that scapular and humeral motion are simultaneously continuous. As this ratio pertains, it is evident that the total range of scapular motion is not more than 60 degrees, nor that of the glenohumeral joint greater than 120 degrees. Under special and abnormal conditions, the motions of either one of these two joints can occur independently. For example, when the scapula is fixed, it is possible to raise the arm actively to the right angle, and passively to 120 degrees (Fig. 11). However, observation and measurement demonstrate that the loss of the effective bone leverage, owing to lack of scapular participation, consequently diminishes power by a third. It should be pointed out in passing that, for free and full elevation of the extremity, lateral rotation of the humerus is essential.

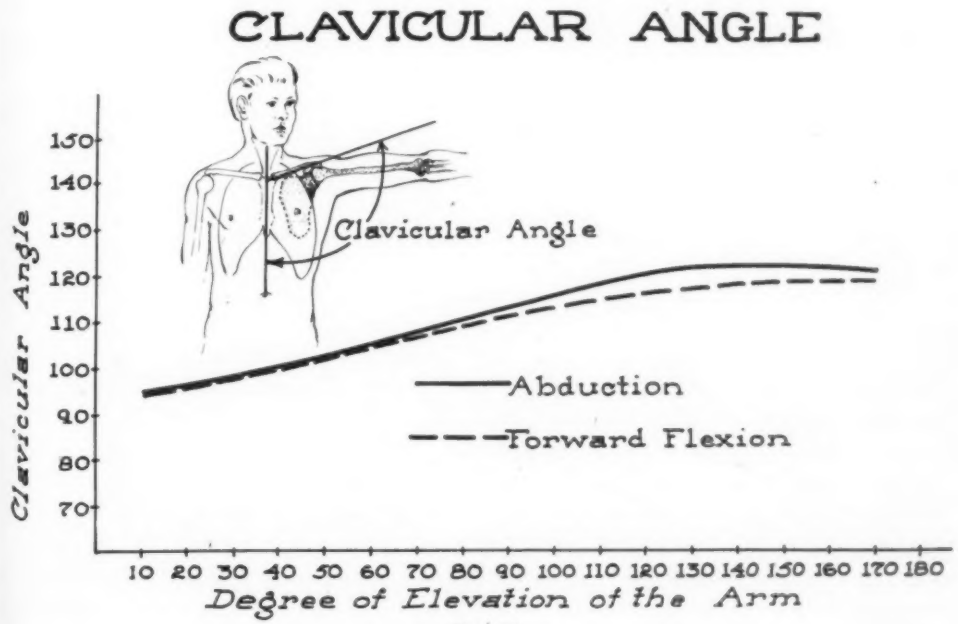


FIG. 12

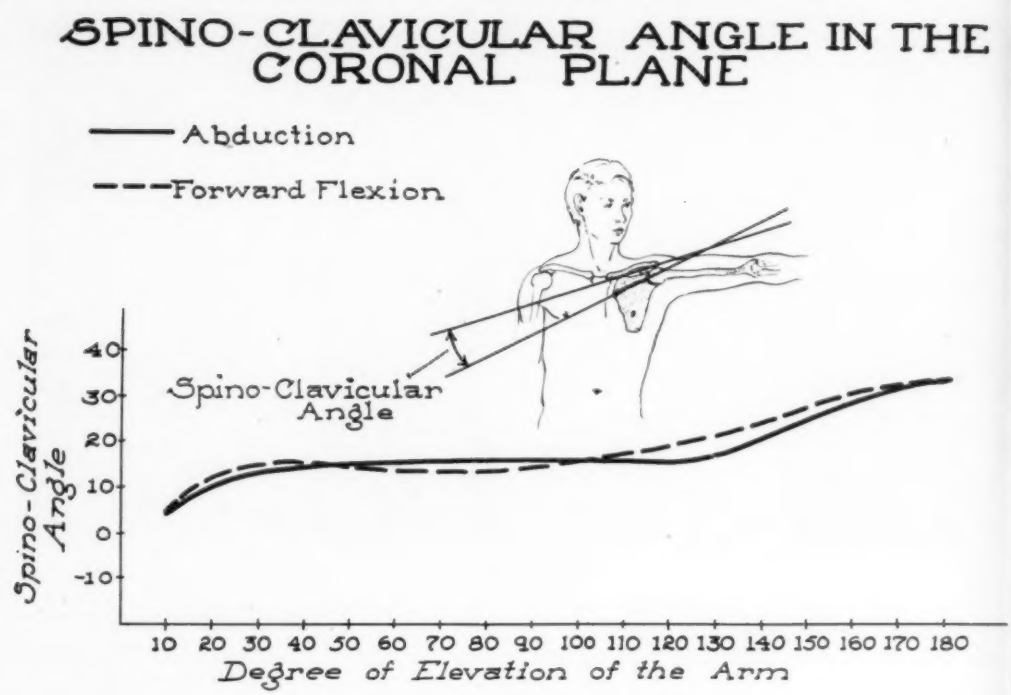


FIG. 13

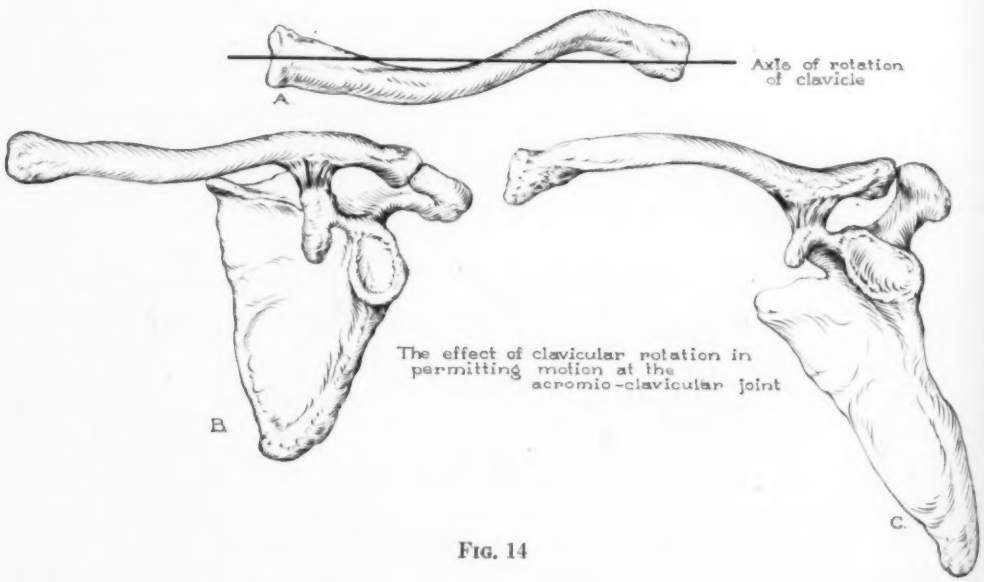

Axis of rotation of clavicle
A
B
C
The effect of clavicular rotation in permitting motion at the acromio-clavicular joint

FIG. 14

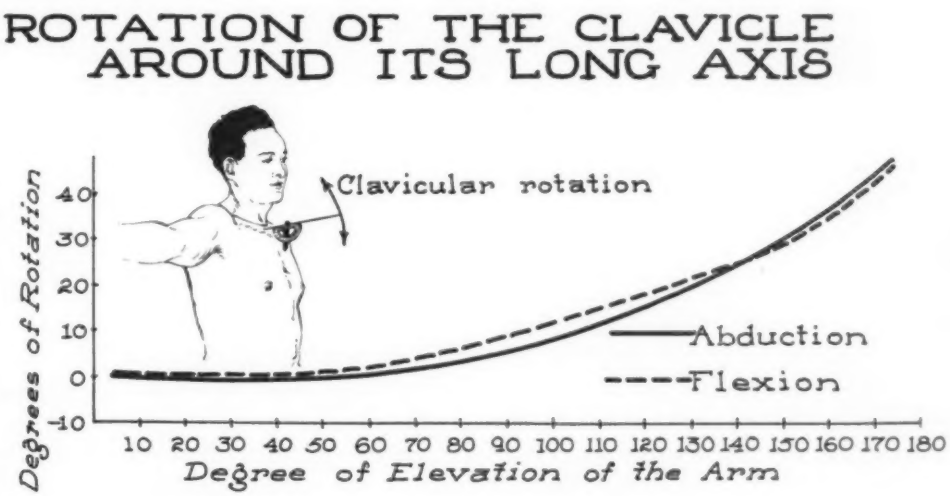
Clavicular motion is more complicated than has been hitherto suspected. The continuous rotation of the scapula on the thoracic wall during elevation of the extremity is only possible because of the motion permitted at the two clavicular joints, and the phase and amount of movement is unequally distributed between them.

Elevation of the arm is accompanied by elevation of the clavicle at the sternoclavicular joint (Fig. 12). This movement begins early and is almost complete during the first 90 degrees, when for every 10 degrees of elevation of the arm, there are 4 degrees of elevation of the clavicle. Above 90 degrees, clavicular motion at this joint is almost negligible.

Motion at the acromioclavicular joint contrasts markedly with that found at the

sternoclavicular joint (Fig. 13). The total range is approximately 20 degrees and occurs both early, in the first 30 degrees of abduction, and late, after 135 degrees of elevation of the arm. Between these two points there is almost no motion of this joint.

The sum of the movements at the sternoclavicular and acromioclavicular joints is naturally equal to the range of movement permitted the scapula, the two bones being welded together by means of their ligamentous attachments. For this reason, it was difficult to understand how motion of such extent could occur at the acromioclavicular joint, in view of the fact that the clavicle is rigidly attached at its lateral extremity to the scapula through the medium of the coracoclavicular ligament. For motion to occur at the acromioclavicular joint in the plane of elevation of the arm, elongation of this ligament would appear to be necessary, and on first sight this would seem to be impossible. Because of the marked curvature of the outer third of the clavicle, we could envisage a relative elongation of the coracoclavicular ligament, only by the clavicle rotating on its long axis, so as to allow this curvature to act as a crankshaft (Fig. 14).



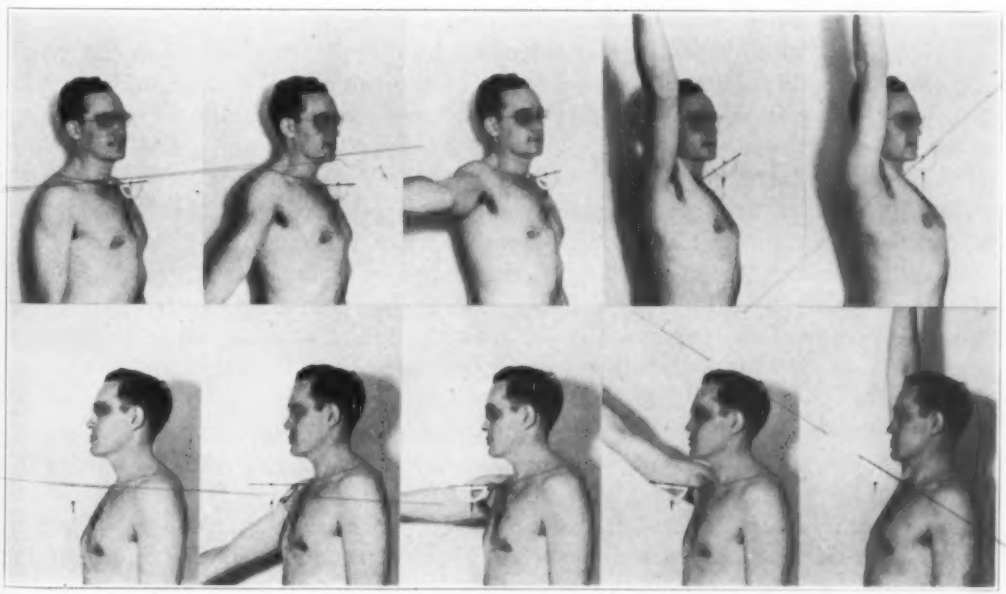


FIG. 15

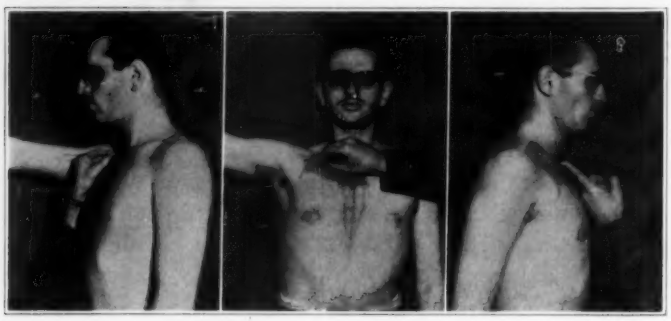


FIG. 16

We were able to measure the excursion of this rotation experimentally by insertion of a pin into the bone of the living subject (Fig. 15). We found that clavicular rotation was a fundamental feature of shoulder motion, for by manually preventing rotation of the pin, the range of motion at the shoulder was restricted to 110 degrees (Fig. 16).

These findings bore out our experience of three years before, when we attempted to treat a case of acromioclavicular dislocation by nailing the joint. We found that, thereafter, this patient was unable to elevate the extremity beyond 60 degrees. We were, therefore, particularly interested in an article by Caldwell in which he discusses a series of cases treated by arthrodesis of the acromioclavicular joint. We would prognosticate that, if fusion of this joint was carried out with the arm elevated to less than 30 degrees, there would be limitation of shoulder-joint motion to slightly below 90 degrees. Whereas, if fusion was carried out with the arm abducted above 30 degrees, the extremity could be carried at least to 135 degrees without difficulty. Even under these circumstances, however, we would expect some limitation of motion between 135 and 180 degrees of elevation.

In addition, knowledge of the significance of this joint suggests the possibility of increasing the functional range of abduction, after arthrodesis of the glenohumeral joint, by excision of the outer end of the clavicle.

MECHANICAL REQUIREMENTS FOR SHOULDER-JOINT MOTION

After measuring by means of roentgenographic studies and direct methods the precise relationships of the bony parts to one another, and the relative positions which they occupy during motion, we were able to set up equations and to calculate the force requirements for the maintenance of the extremity during flexion or abduction. By the technique employed, these calculations were arrived at by theoretical analysis, and the results were then checked on a force model which yielded empirical data.

To establish equilibrium at the glenohumeral joint in any position of the arm, a minimum of three forces is required. One of these is represented by the weight of the extremity, acting at the center of gravity of the limb; the second, the abducting musculature represented predominantly by the deltoid; and the third, the resultant of the former forces acting through the center of rotation in a direction opposite to that of the deltoid (Fig. 17). This last force is, in turn, obviously the resultant of other components, for there is no muscle capable of exercising pull in the direction which it occupies. These components are undoubtedly: first, the pressure and friction of the head of the humerus at the glenoid; and, second, the downward pull of muscles—such as the subscapularis, infraspinatus, and teres minor—whose action is below the center of rotation of the humeral head. In order to represent them, we therefore selected two forces, one acting at right angles to the plane of the glenoid, and the other, parallel to the axillary border of the scapula.

The theoretical and empirical values for these forces were determined in the range from 30 degrees to 180 degrees of elevation, as below 30 degrees individual variation is so great as to make the findings highly questionable (Fig. 18).

The curve representing the force requirements for elevation, almost a pure sine wave, reaches its summit at 90 degrees of elevation, where the magnitude of the force required represents 8.2 times the weight of the extremity. It then falls progressively to zero at 180 degrees, because, when the extremity is in the exact vertical position above the head, no

force is theoretically required to maintain that position. In the living mechanism, muscle power is expended throughout the full range of motion. The second force is the resultant which we have broken up into two components representing, on the one hand, active force in the form of the downward pull of infraspinous muscles and, on the other, the passive resistance of pressure and friction. The active component, shown in myographic recordings of the muscles during motion, is one of the greatest significance and importance. It establishes, with the pull of the deltoid, the essential force couple necessary for elevation of the extremity. This force reaches its maximum at 60 degrees, where its magnitude is 9.6 times the weight of the extremity*, a pull greater than that of the elevating force. Beyond 90 degrees the force falls rapidly, reaching zero at 135 degrees.

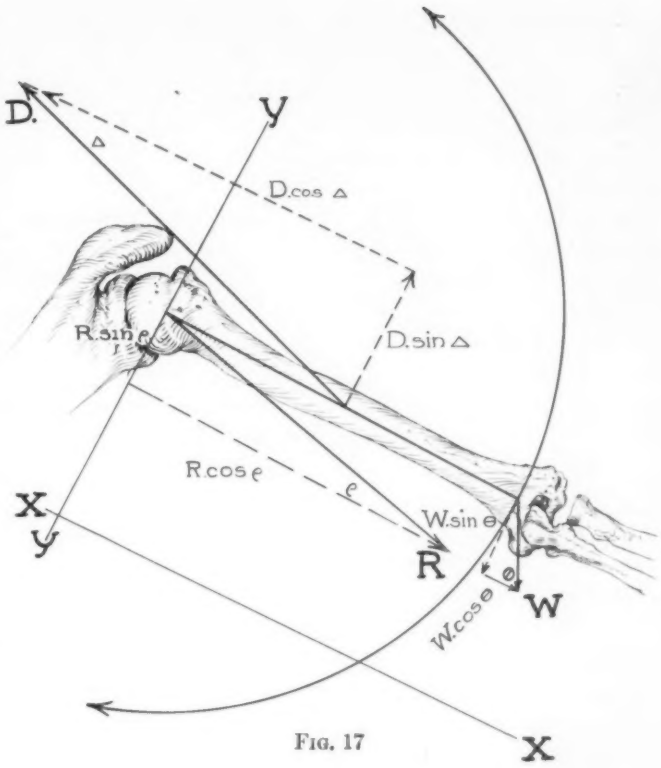


FIG. 17

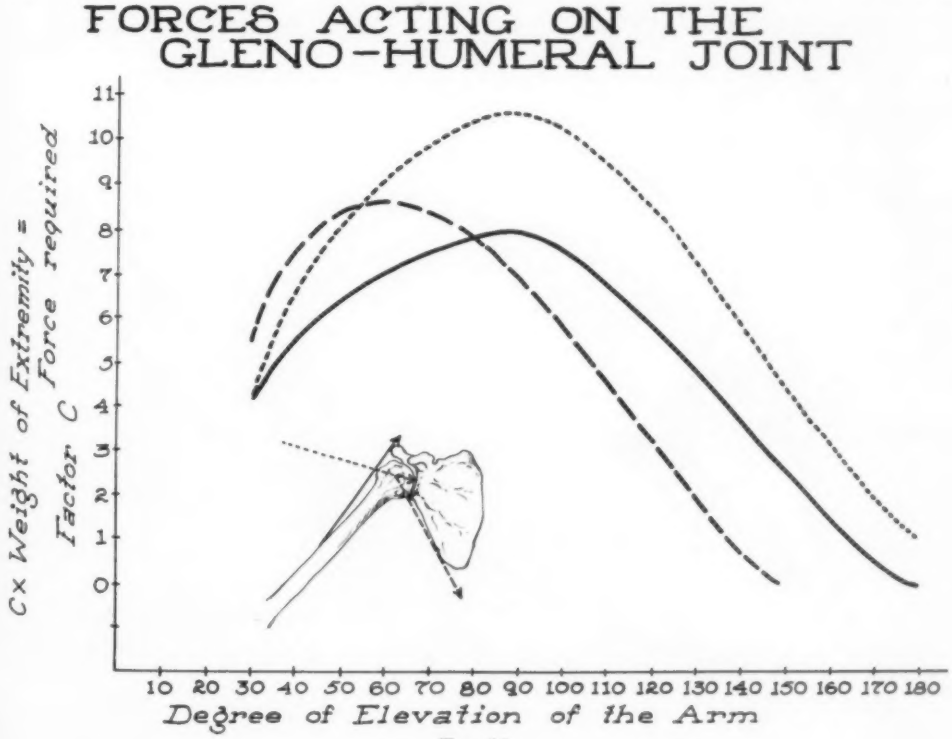


FIG. 18

*The weight of the upper extremity, amputated at the glenohumeral joint, is approximately 9 per cent. of the total weight of the body.

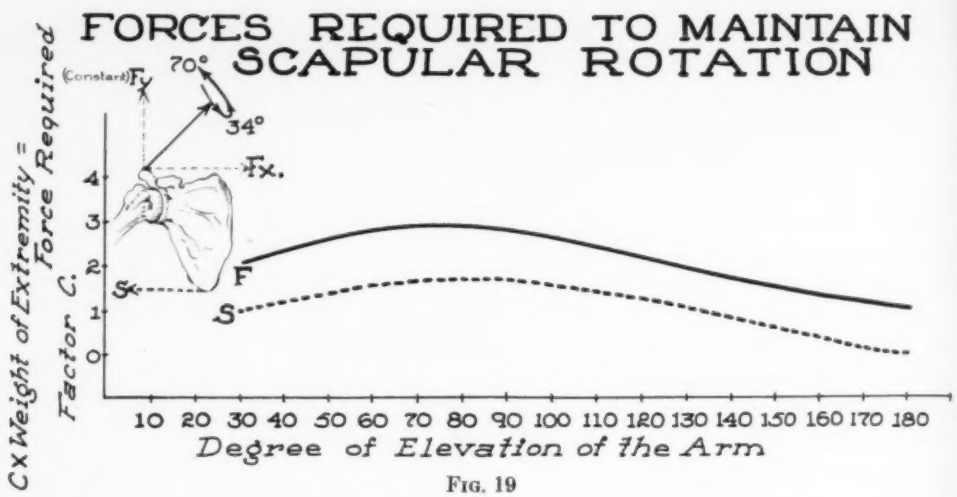


FIG. 19

The pressure and friction against the glenoid is a force of considerable size, which reaches its summit at 90 degrees of elevation, where it represents no less than 10.2 times the weight of the extremity.

We should like to emphasize the importance of the muscle force couple as an essential principle in the mechanics of elevation, as this same principle will be seen to operate in scapular rotation. Here, as at the glenohumeral joint, three essential forces participate in the mechanism (Fig. 19). The first of these is the force necessary to counteract the weight of the entire shoulder girdle which, therefore, acts in a vertical and upward direction. The

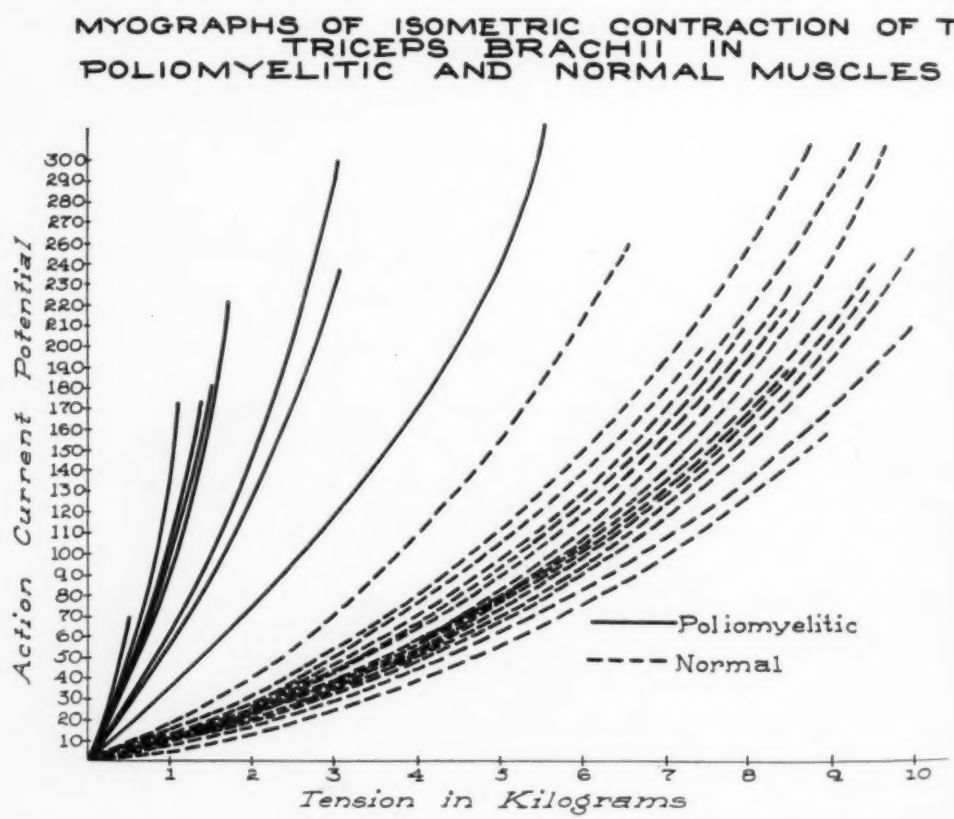

MYOGRAPHS OF ISOMETRIC CONTRACTION OF THE TRICEPS BRACHII IN POLIOMYELITIC AND NORMAL MUSCLES
Action Current Potential
10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180 190 200 210 220 230 240 260 270 280 290 300
1 2 3 4 5 6 7 8 9 10
Tension in Kilograms
Poliomyelitic
Normal

FIG. 20

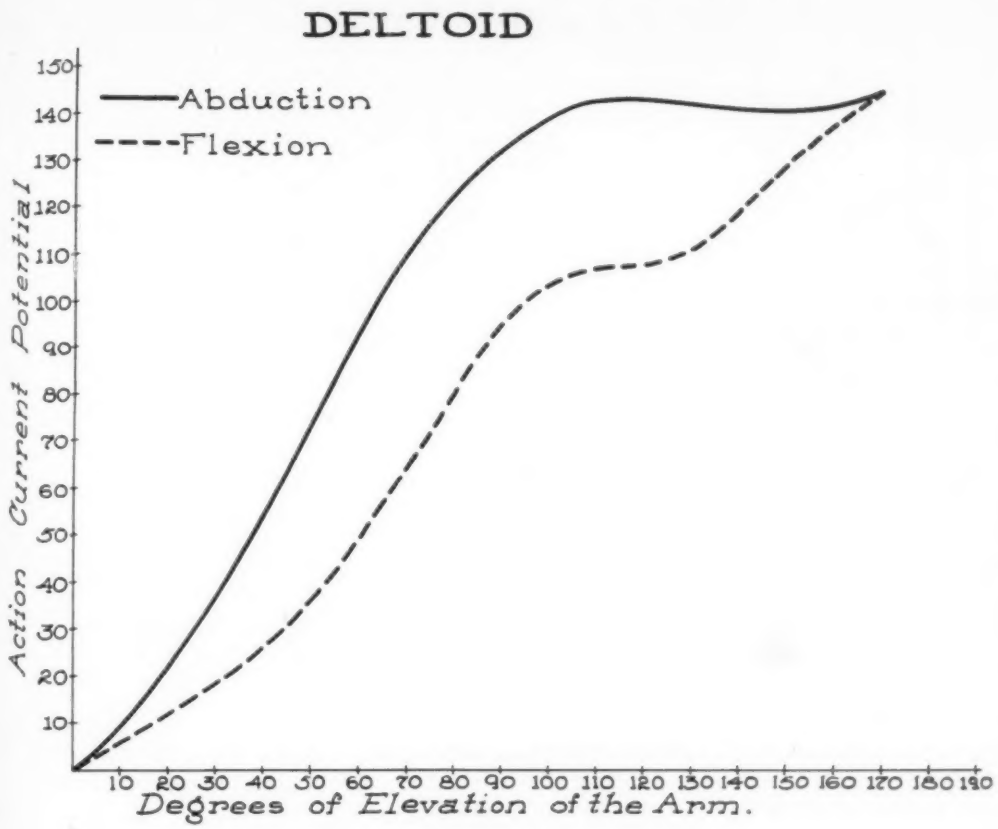

DELTOID
Abduction
Flexion
Action Current Potential
150
140
130
120
110
100
90
80
70
60
50
40
30
20
10
10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180 190
Degrees of Elevation of the Arm.

FIG. 21

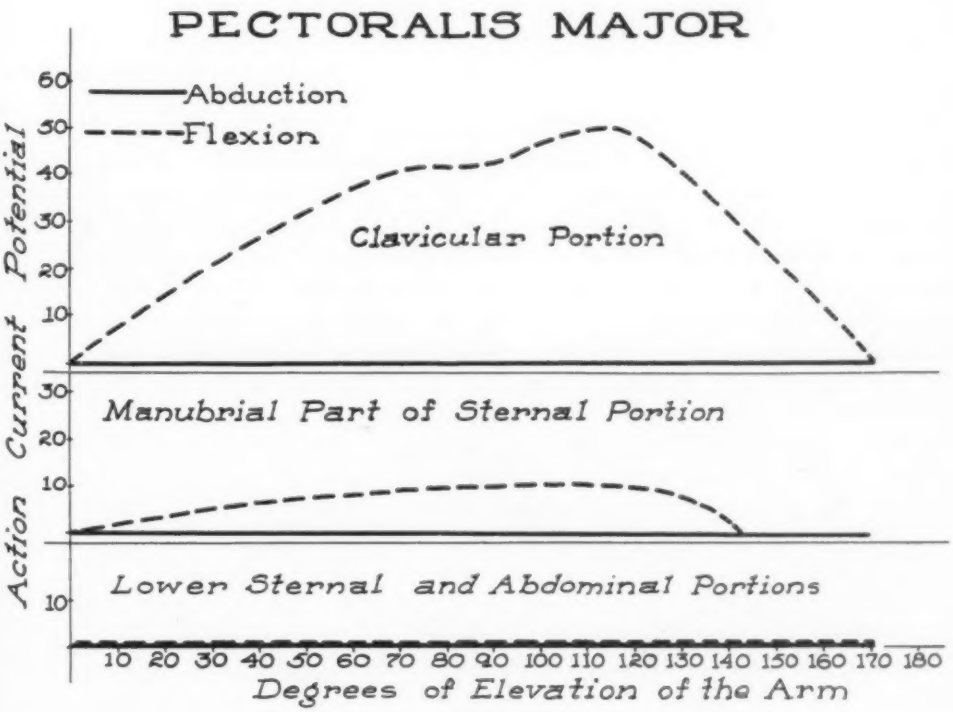

PECTORALIS MAJOR
Abduction
Flexion
Action Current Potential
60
50
40
30
20
10
Clavicular Portion
30
20
10
Manubrial Part of Sternal Portion
10
Lower Sternal and Abdominal Portions
10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180
Degrees of Elevation of the Arm

FIG. 22

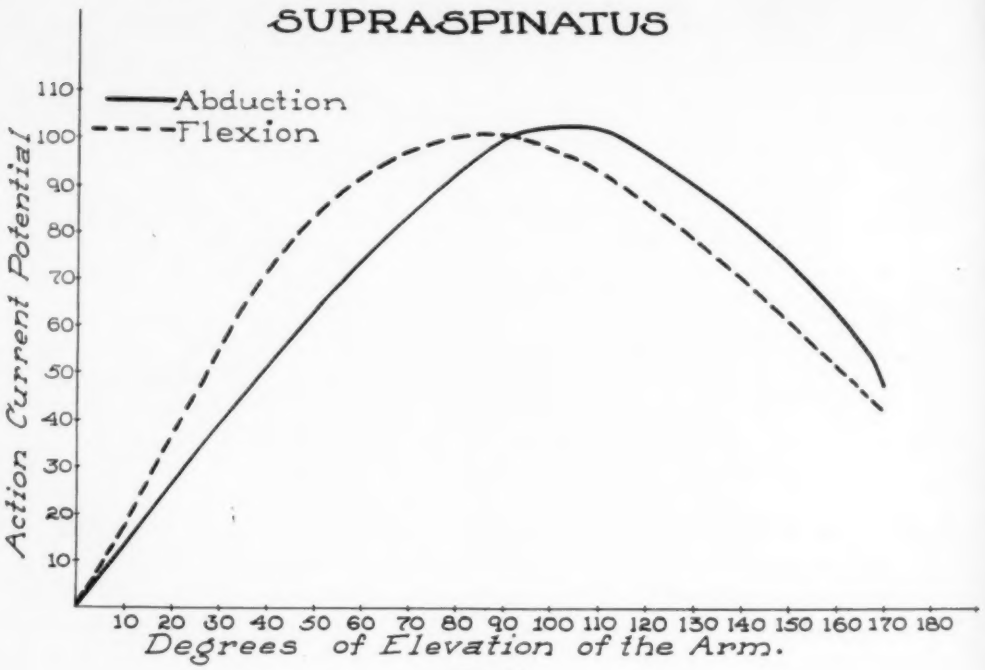


FIG. 23

other two forces establish the rotary couple, one acting from the region of the acromion process in a medial direction, and the other from the inferior angle in an outward or forward direction. The force acting on the inferior angle is represented predominantly by the serratus anterior. That acting on the acromion is in part passive, and in part active. The passive component is resisted by the antagonistic pressure of the clavicle. The active or kinetic component is supplied by the upper portion of the trapezius, which must, however, also, act in supporting the weight of the pectoral girdle. The force acting from the acromion is a resultant, fulfilling the requirements of both the supportive and the rotary rôles.

The weight of the extremity is a constant during the entire range of motion, and is consequently represented by a straight line. The forces necessary to establish the upper and lower components of the rotary force couple are equal but opposite in direction, and reach their summit at 90 degrees of elevation, theoretically falling to zero at 180 degrees. Owing to the changes in position of the scapula during elevation, the resultant of the supportive and rotary components supplied by the upper portion of the trapezius was found to fluctuate in its angle of action. This fluctuation reveals an interesting mechanism in the action of the trapezius. In the resting position, the muscle is entirely supportive. With the first 35 degrees of elevation, the angle of action of the muscle changes, so that its force is equally divided between the supportive and the rotary rôles. From 35 to 140 degrees, the muscle is increasingly more effective as a rotator, with its maximum power at 90 degrees, and beyond 140 degrees its rotary efficiency decreases and its supportive rises. This complicated mechanism of action of the upper trapezius is achieved by simple elevation of the shoulder girdle as a whole. In consequence, where the supportive rôle is the more important, the muscle acts predominantly for this purpose. As rotation of the scapula develops, progressive changes in the angle of action of the muscle enable it to serve the requirements of the rotary couple more effectively.

The magnitude of the forces rotating the scapula, in contrast to those acting on the glenohumeral joint, is very much smaller. The forces reach their maximum at about 90 degrees, when they are approximately only twice the weight of the extremity.

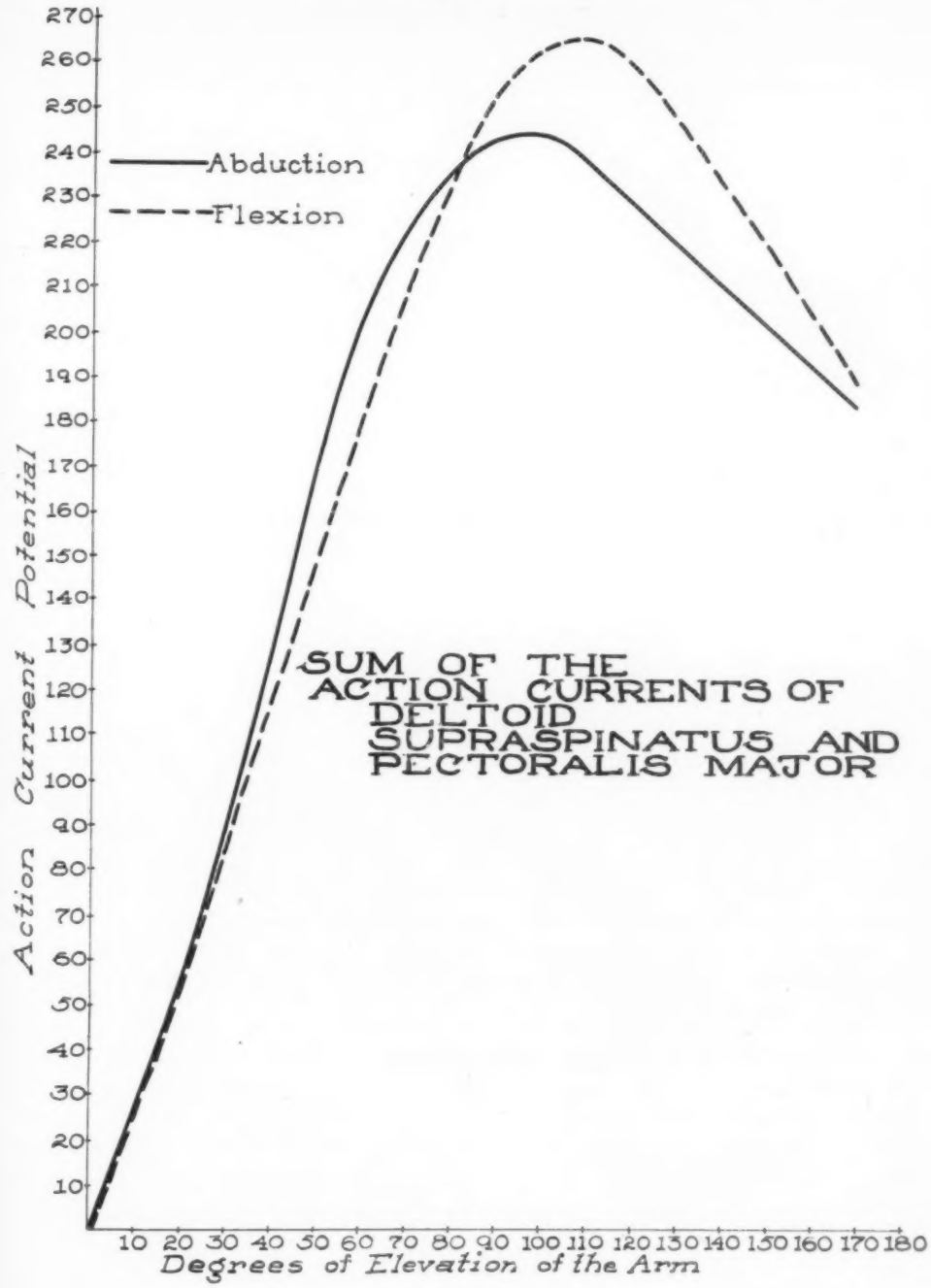


FIG. 24

MYOGRAPHIC STUDIES

In order to correlate the findings and test the validity of the conclusions reached in the comparative anatomical studies and by analysis of the mechanical-force requirements of the shoulder mechanism, we have employed an entirely new method of approach. The method consists in sampling the muscle activity developed during motion in the living subject. The electrical action potentials of the muscle are drawn off through electrodes, implanted in their substance. The differences in potential so obtained are amplified and mechanically recorded. The procedure is valuable, not only for analyzing the activity of

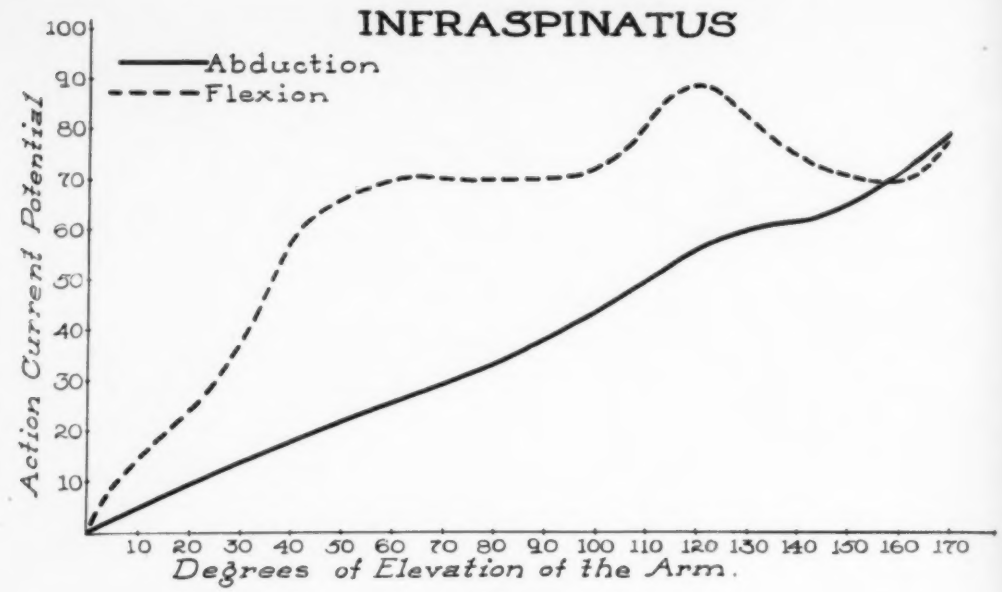


FIG. 25

an individual muscle during motion, but also for the study of the phase of action of muscle groups participating in any free coordinated movement. The use of six separate amplifiers has enabled us to examine the action of a like number of muscles simultaneously.

Experiments have determined that a direct relationship between the tension developed in a muscle and the action current potential, as recorded by the magnitude of the amplitude, exists (Fig. 20). This relationship is not however a linear one, for in normal muscle the action potential rises more rapidly than the increase in tension, the precise relationship being the function of the square, and is expressed as a quadratic equation. It is interesting to observe that muscles affected by poliomyelitis behave somewhat differently. In postpoliomyelitic paralysis with increasing tension, the action potential rises far more rapidly than in the normal. The shape of the curve is changed and the relationship now becomes logarithmic. We have not been able as yet to establish the significance of these changes in the abnormal muscle.

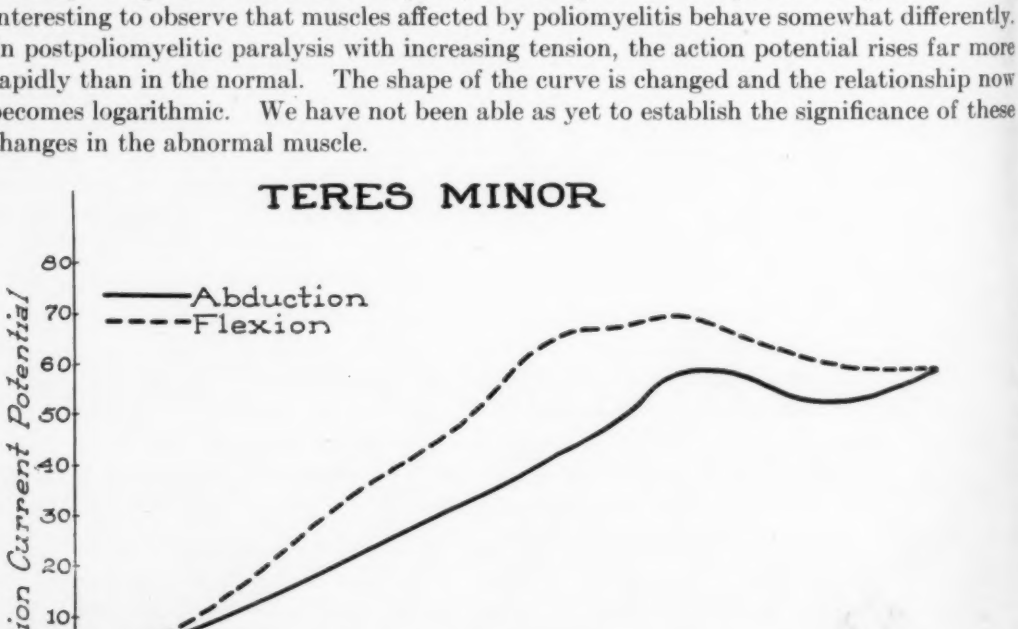


FIG. 26

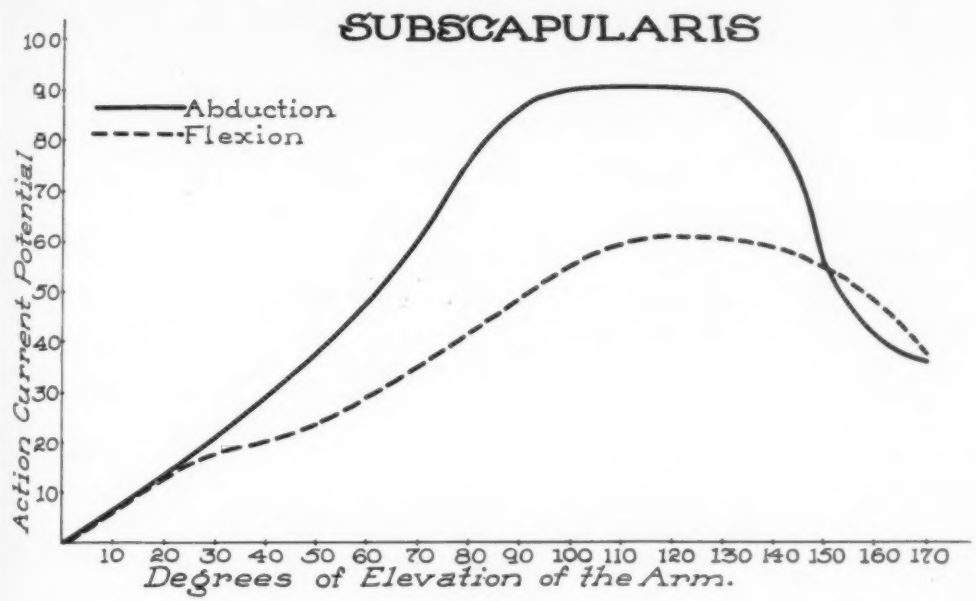


FIG. 27

As the sampling of muscle depends upon the distance between the inserted electrodes, we are thus able to record the activity either of the entire muscle or of any of its parts. This is an important asset, because it reveals that different portions of the same muscle differ in their degree of activity, depending upon the precise motion carried out.

From the records of their action potential and phase of activity, we have been able to construct, based upon averages from four to twenty individual observations, a series of curves for each muscle participating in the shoulder motion. Analysis of these reveals that we must group the muscles into functional rather than into topographical groups.

The Abductors and Flexors of the Humerus

The Deltoid: The deltoid muscle exhibits its greatest activity between 90 and 180 degrees of elevation, the curve plateauing between these points (Fig. 21). In flexion, the total potential is of less amplitude than in the corresponding curve for abduction. It reaches an initial peak at 110 degrees, plateaus to 130 degrees, and finally rises abruptly to attain the same level as that for abduction when the arm is fully elevated above the head.

Pectoralis Major: The pectoralis major exhibits differences in activity in the various portions of the muscle, and these in turn vary with the type of motion carried out (Fig. 22). In abduction no portion of the pectoralis major is active. In forward flexion, the clavicular head is most active, reaching a primary peak at 75 degrees, and a secondary peak at 115 degrees. The manubrial segment of the sternocostal head is the only other part of the muscle to show any activity in flexion. Its activity is, however, slight; it is maximum at 110 degrees, and, thereafter, falls abruptly to zero at 145 degrees. The lower sternal and abdominal portions are inactive in both flexion and abduction. These studies clearly reveal that the clavicular head of the pectoralis major works synchronously with the anterior deltoid in forward flexion, and accounts for the relatively low level of the action potential of the deltoid found in this movement.

Supraspinatus: The curve for the supraspinatus describes almost a pure sine wave, which is strongly reminiscent of the force curve elaborated by mechanical analysis (Fig. 23). In forward flexion it reaches its peak slightly before that of the abduction curve, the maximum being at 80 degrees, while that of abduction is greatest at 100 degrees. The heights of the curves in both flexion and abduction are the same. It is commonly considered

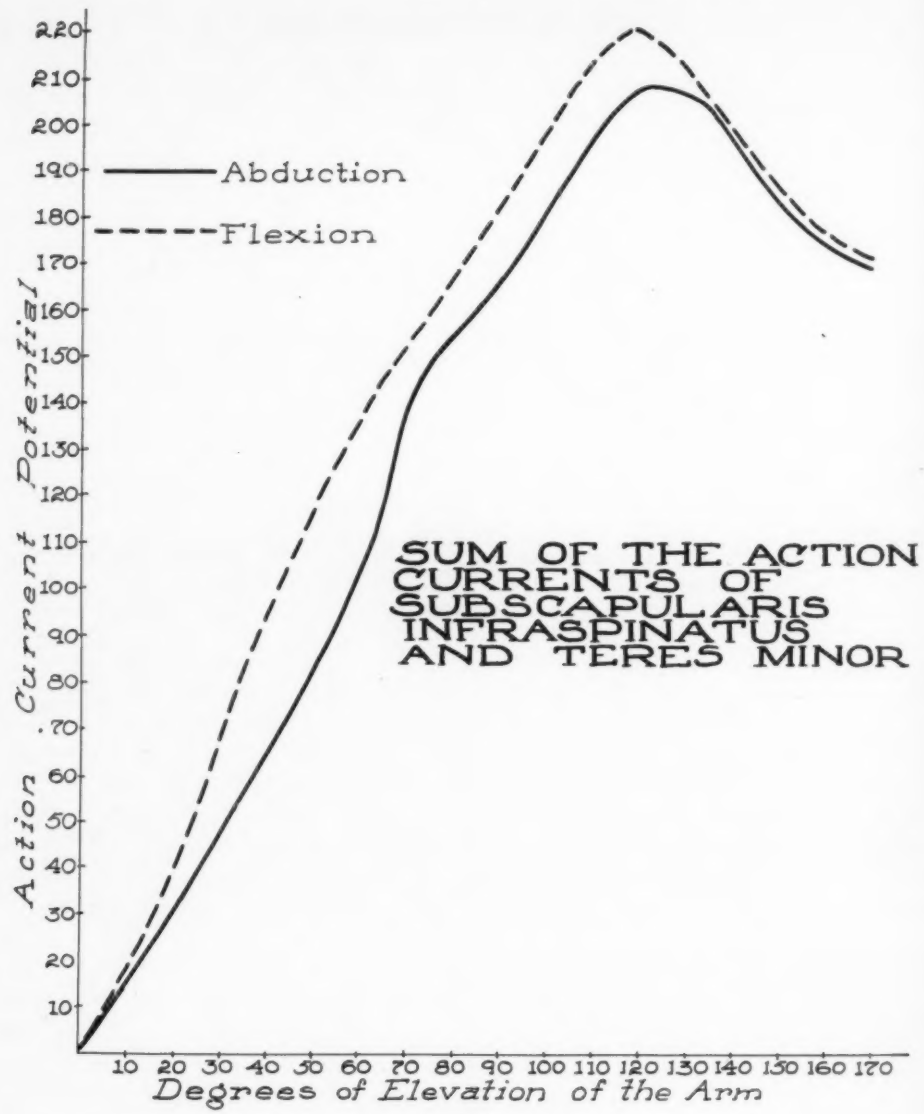

Abduction
Flexion
SUM OF THE ACTION CURRENTS OF SUBSCAPULARIS INFRASPINATUS AND TERES MINOR
Action Current Potential
Degrees of Elevation of the Arm
10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170
10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180 190 200 210 220

FIG. 28

that the supraspinatus is the initiator of abduction. The action-potential curves, however, definitely show that this is not the case. The muscle acts together with the deltoid progressively throughout the entire range of motion.

When we summate the action potentials of all the abducting musculature, we find that individual irregularities disappear, and a smooth curve results which compares very closely with that established from analysis of the force requirements for elevation of the arm (Fig. 24). In abduction, the summit is reached at 90 degrees and flexion slightly later at 110 degrees. It will be noted that the flexion curve is of somewhat greater amplitude than that of abduction, and further is displaced slightly to the right. We would account for the difference in amplitude in flexion as being due to the activity of the clavicular head of the pectoralis major, whose force is utilized in this movement, not only for elevation, but partly to maintain the forward position of the arm. The additional functional requirement would account for the slight increase of amplitude of the curve and its displacement to the right when compared to the force-requirement curve. Muscle activity does not fall to zero at 180 degrees of elevation, indicating that, unlike the theoretical calcula-

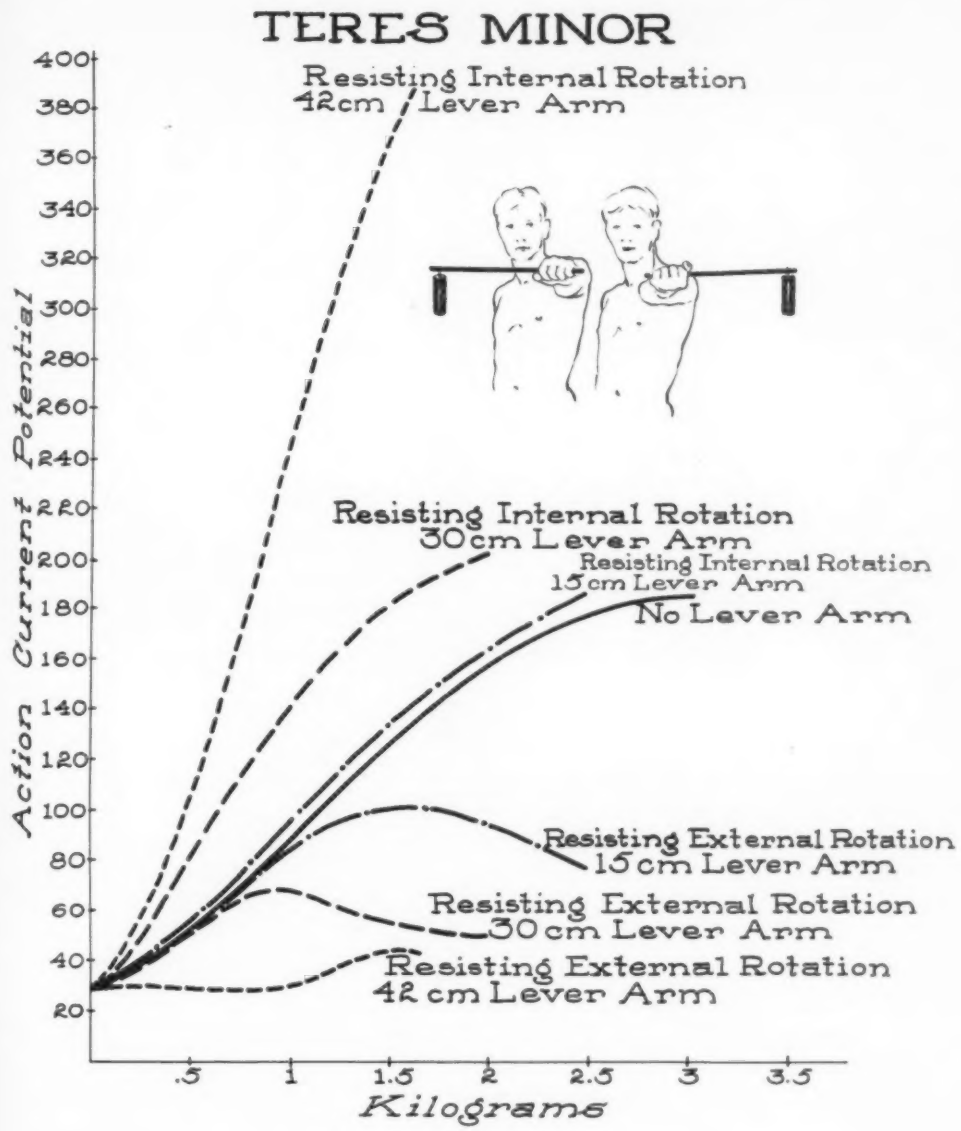


FIG. 29

tions, muscle activity is still required, as is to be expected, to maintain the extremity above the head. However, in this position their amplitude is reduced.

The Depressors of the Humerus

The subscapularis, infraspinatus, and teres minor constitute a functional group which, as already revealed from the force studies, act as the second or inferior component of the force couple. These muscles were, therefore, found to act continuously throughout both abduction and flexion.

In abduction, activity of the infraspinatus rises in almost linear fashion to attain its summit at 180 degrees. In flexion, the curve is somewhat higher, is irregular, and exhibits two major peaks of activity, the first at 60 degrees, and a second of greater amplitude at 120 degrees. Thereafter the curve falls to coincide with the abduction curve (Fig. 25).

The teres minor behaves in a similar fashion to that of the infraspinatus (Fig. 26). The abduction curve is almost linear, but slightly peaked at 120 degrees of abduction. In

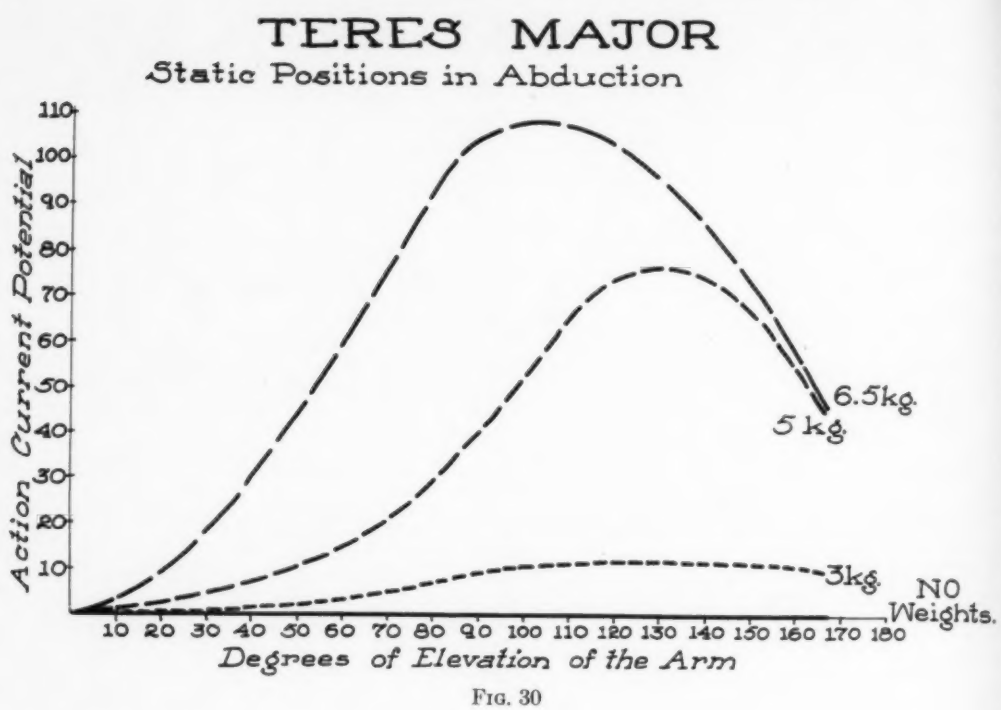


FIG. 30

flexion, the total amplitude is higher, rising to its maximum between 90 and 120 degrees, thence falling slightly to coincide with the curve of abduction.

The curve of activity of the subscapularis contrasts with those of the infraspinatus and teres minor in that the picture is reversed (Fig. 27). In this muscle the abduction curve is of higher potential than the flexion, and reaches its summit at 90 degrees, is maintained from there to 130, and thence falls abruptly to 180. The amplitude of the flexion curve is lower, and its summit is arrived at later—between 110 and 130 degrees—and thereafter coincides with that for abduction.

If we summate the amplitudes of these three depressor muscles, we find that on the whole they describe a curve remarkably similar to that of abduction, excepting that the summit of the curve occurs a little later,—namely, at 110 to 120 degrees, the flexion curve being slightly higher in amplitude than that of abduction (Fig. 28). There is, however, an important and remarkable secondary peak prominent in the curve for abduction which is maximal between 60 and 80 degrees. This secondary peak occurs at the level which force analysis indicates is the period when the depressor action of the lower force couple should be most active. We believe that this curve is formed by the superimposition of two essential forces which are necessary for abduction. The first peak is unquestionably due to the depressor action and the second to the activity of those muscles in their function as rotators. That this interpretation is correct is shown by the behavior of these muscles when the rotary action is minimized by the addition of a lever and weight so as to resist this movement in one or the other direction (Fig. 29). We find for example that we are totally unable to suppress the activity of these muscles by passively supplying the rotary power through the medium of weights applied to varying lengths of lever arm. The more we increase rotary resistance with such apparatus, the more we weaken the power of abduction. Although in these experiments, we have in no way altered the power of the abducting musculature, nonetheless, by absorbing an increasing amount of the depressor power of the muscle for the purposes of rotation, we have weakened the lower element of the couple, and in consequence minimized its effectiveness for abduction.

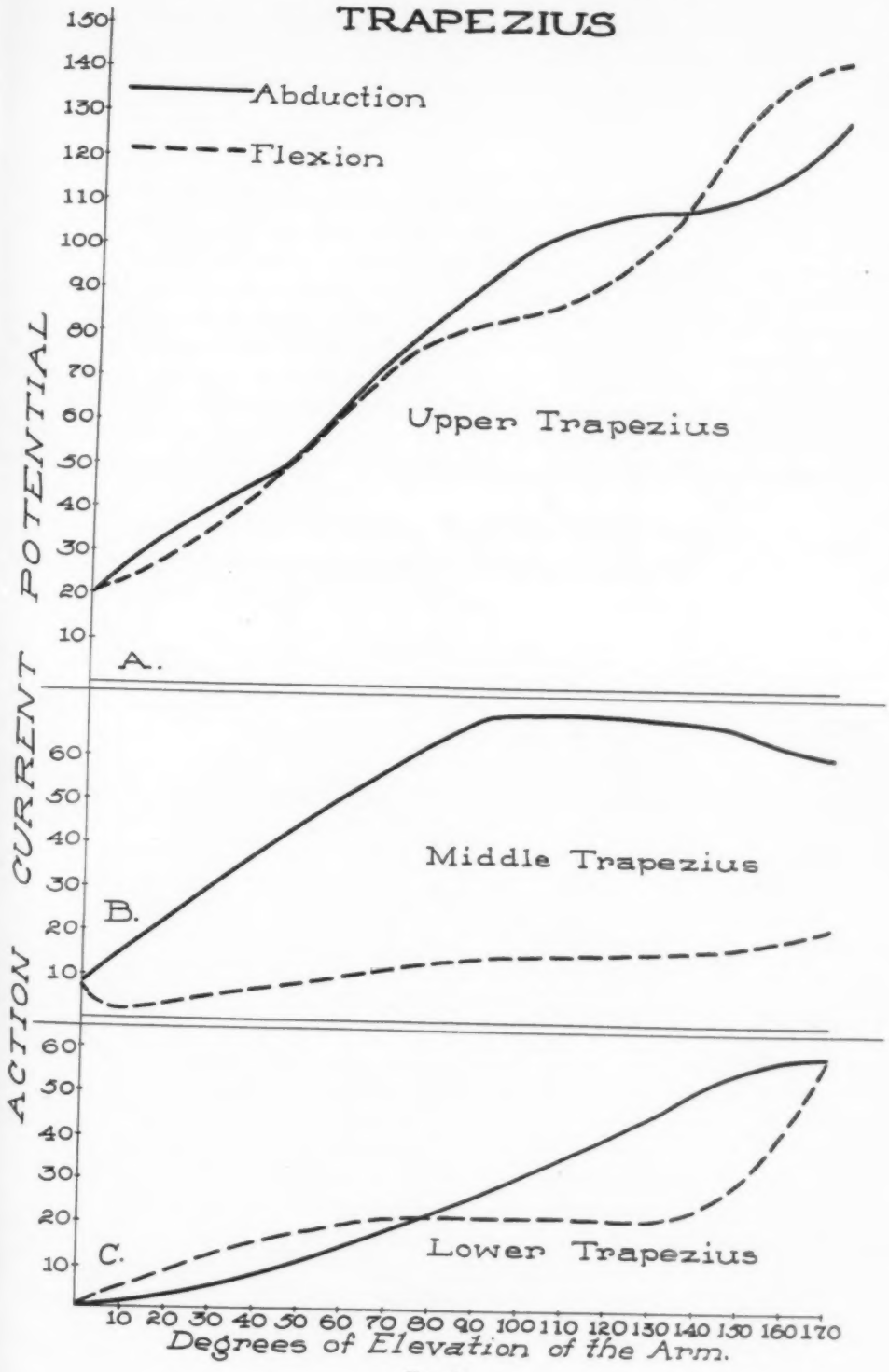


FIG. 31

Teres Major

The teres major occupies a special position in the scapulohumeral musculature. The muscle never exhibits any activity during motion, but plays a peculiar rôle in that it only comes into action when it is necessary to maintain a static position (Fig. 30). In static

positions, it reaches its maximum activity at about 90 degrees. The teres major, in elevation of the extremity, therefore, is not a kinetic muscle, but is important in maintaining a static posture, its activity directly increasing with the increase of weight.

The Scapular Rotators

The scapular rotators are a distinct functional group. We have already pointed out from comparative anatomical studies that both the trapezius and serratus anterior show evidence of division into separate upper, intermediate, and lower components. This anatomical separation is reflected in the functional activity of these portions of the muscles.

The upper trapezius, levator scapulae, and upper digitations of the serratus anterior constitute a unit whose activities are essentially the same. The unit performs three functions: passive support of the shoulder, active elevation of the shoulder, and, in addition is the upper component of the force couple necessary for scapular rotation. The postural function of the group is evidenced by the fact that these muscles exhibit an action current potential while the arm is at rest (Fig. 31). With elevation of the extremity, both in flexion and abduction, the amplitude of their action current potential rises in linear form,

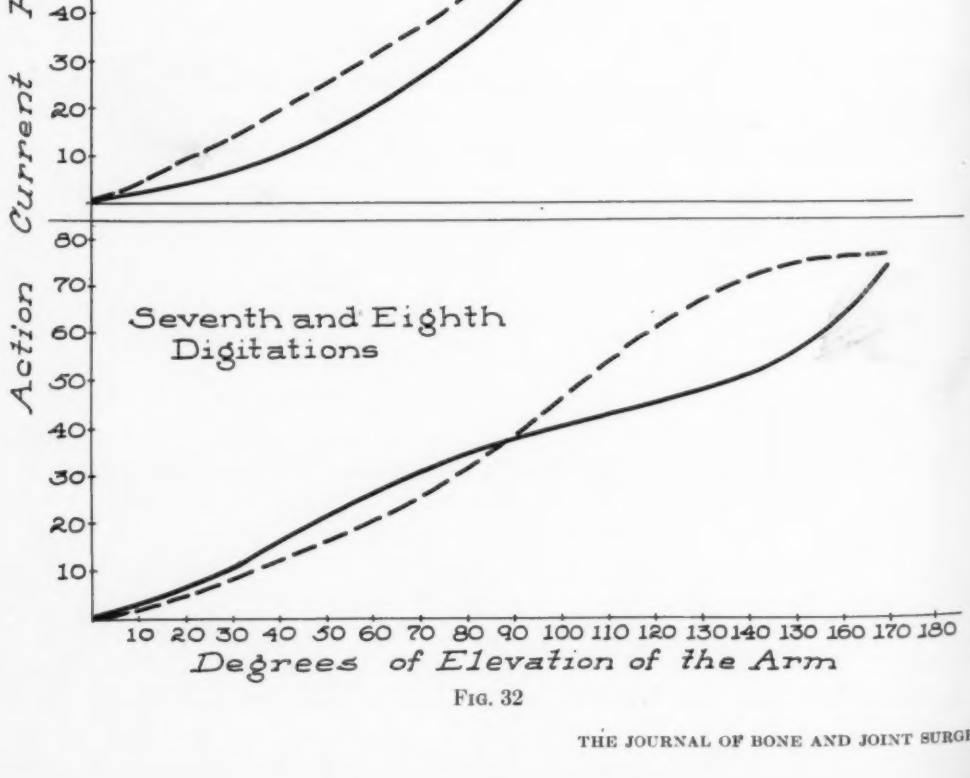


FIG. 32

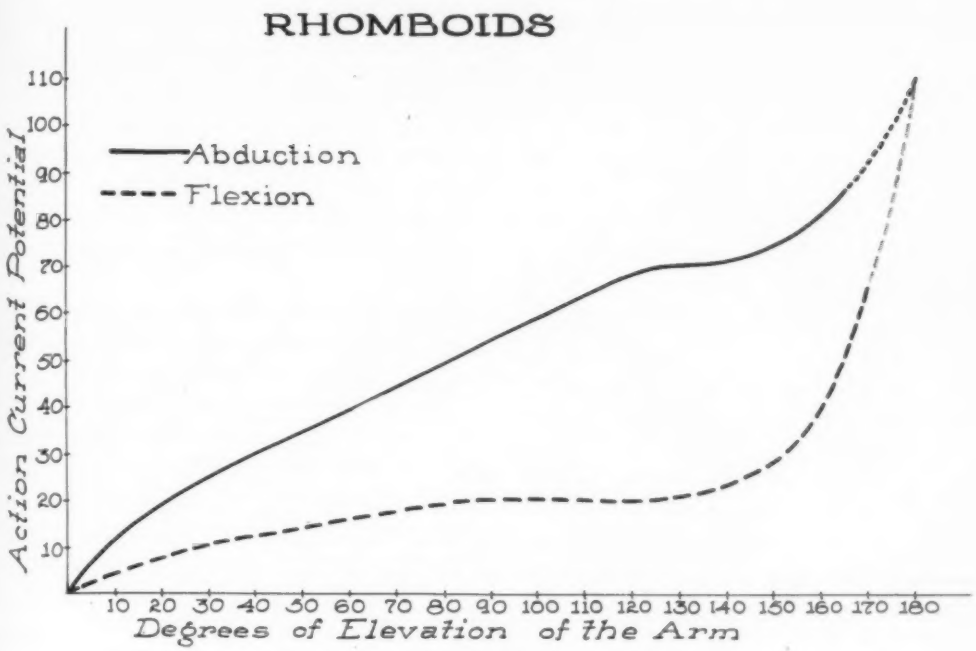


FIG. 33

reaching its maximum when the arm is above the head. The curve undulates slightly above 90 degrees, for mechanical analysis has shown that the supportive function of the muscle at this point becomes one predominantly associated with rotation of the scapula.

The inferior portion of the trapezius and the lower four digitations of the serratus anterior constitute the lower component of the scapular rotary force couple, and are found to act throughout elevation of the extremity in a complementary manner (Fig. 31*C* and Fig. 32). The general shape of their action curves is similar, starting at zero and reaching their maximum at 180 degrees of elevation. The inferior portion of the trapezius and the seventh and eighth digitations of the serratus anterior describe curves which are mirror images of one another. The inferior trapezius acts with slight undulations continuously and predominantly in abduction, the seventh and eighth digitations of the serratus predominantly in flexion. The curves demonstrate, therefore, that the lower trapezius is the more active component of the lower force couple in abduction, for in flexion it relaxes somewhat to allow the scapula to migrate forward as a whole, and the major component of the lower force couple is then supplied by the lower digitations of the serratus anterior.

The intermediate portion of the trapezius muscle is most active in abduction, when its potential rises to a maximum at 90 degrees, flattens off, and finally falls slightly at 180 degrees (Fig. 31*B*). In forward flexion, its action potential decreases in amplitude during the early ranges of movement, and then builds up slightly to 180 degrees. The findings indicate that the middle trapezius functionally serves to fix the scapula in its plane of motion during abduction, and relaxes somewhat in forward flexion to allow the scapula to rotate around the thorax.

The rhomboid muscles function in much the same manner as the middle trapezius, and, like it, are most active in abduction (Fig. 33). In flexion, their amplitude is found to flatten off between 60 and 150 degrees, after which it rises sharply to its maximum at 180 degrees. The curves indicate that the rhomboid and the middle trapezius are but little active in flexion. Once, however, 140 degrees is reached, a position little different whether the arm has been carried to that level by flexion or by abduction, the muscles strongly contract and the flexion and abduction curves superimpose.

SUMMARY AND CONCLUSION

The eclectic approach to an understanding of the functional mechanism of the shoulder enables us to lay down, with considerable certainty, fundamental principles which are of supreme significance to the surgeon in planning reconstructive procedures for both postpoliomyelitic paralysis and other disorders affecting this region. The evidence obtained from comparative anatomical trends, the changes in the relative size of the musculature, the elaboration of the bony parts, the theoretical force requirements, and the basic behavior of the muscles in the living, as revealed by myographic analysis, is consistent, and points in the same general direction.

Briefly, the morphological changes have consisted of elaboration and massive development of the deltoid, with a corresponding suppression of the supraspinatus and biceps muscles as active abductors. Associated with these changes, the muscles lying below the spine of the scapula and acting on the humerus assume increasing importance; by establishing the necessary components of a force couple, they, together with the deltoid, bring about the rotary motion necessary for elevation. The upper trapezius, the upper serratus, and levator angulae scapulae, by functional and morphological separation from the lower trapezius and lower serratus, have produced the components of a similar force couple acting upon the scapula. These changes have been attained structurally in surprisingly simple fashion. Elongation of the inferior angle of the scapula, together with minor migrations of muscle masses, such as the teres minor, has accomplished a twofold object in forming the depressor group of the glenohumeral joint, and at the same time in increasing the lever arm for the action of the lower trapezius and serratus muscles as a rotary unit for the scapula. Alterations, such as the distal migration of the insertion of the deltoid and the increasing mass of the acromion, are relatively minor in comparison to the effects achieved by this extension of the scapula, and should be looked upon as adjustments perfecting the essential mechanism.

The studies of joint motion prove conclusively that complete elevation of the arm, in either the coronal or frontal plane, is dependent on free motion in all the joints of the shoulder complex. The old teaching, still commonly accepted, that abduction to the right angle takes place entirely at the glenohumeral joint, and that, thereafter, full elevation is completed by motion of the scapula on the chest wall, is incorrect. On the contrary, it should be stressed that motion occurs in all the joints of the region simultaneously, each contributing its share to the completion of the movement. The maintenance of the rhythm of smooth and coordinated motion requires intact joints and the preservation of power in the muscles that move them.

We may say in summary, that during early phases of elevation of the arm, the sternoclavicular joint passes through its greatest ranges of movement, and in the terminal phase, the acromioclavicular. At the glenohumeral and scapulothoracic articulations, the ratio, from almost the beginning to the termination of the arc, is respectively two to one, so that for every 15 degrees of elevation, the glenohumeral contributes 10 degrees, and the scapulothoracic 5 degrees.

It is helpful in diagnosis to recognize that disturbance of rhythm or actual loss of motion in any one phase of motion may indicate that this disturbance is due to loss of function at the joint or joints which contribute the major share of movement during that phase. Furthermore, ankylosis of any one of the joints associated in the complex will cause a permanent loss in degree of movement in direct proportion to the amount of movement contributed by that joint. In fusion of the acromioclavicular joint, as practiced by some surgeons for its dislocation, a permanent loss of abduction in greater or less degree in the terminal phases of this movement, should be anticipated. Restriction at the sternoclavicular joint will not only limit abduction, but will greatly diminish its power. When the surgeon performs an arthrodesis of the glenohumeral joint, he should be aware that the maximum range is but 60 degrees of actual motion. Evidence, that awaits further ex-

perimental confirmation, suggests that this range might be increased by resection of the outer end of the clavicle with preservation of the coracoclavicular ligaments.

From a functional standpoint, the establishment of force couples at both the scapulothoracic and glenohumeral articulations, is a principle of first importance, and necessitates revision of our former ideas concerning the mechanism of movement at both joints, and, therefore, requires the formulation of new procedures for surgical reconstruction of the paralyzed. Reconstruction necessitates rearrangement of musculature rather than purely empirical substitution of a paralyzed by an unparalyzed muscle. In the past, the orthopaedic surgeon has thought of the movement of abduction in terms of the power of the deltoid. On the other hand, the new concept, herein presented, emphasizes the position of the short rotators which, as depressors, assume equal importance in the essential couple. Without them the deltoid is helpless to move the arm away from the side. If the lower component of the force couple contributed by these muscles is weak and ineffective, transplantations of muscles to substitute for the deltoid are doomed to failure, for they can but serve to translate the humerus in its long axis, impinging the tubercules and head of the bone against the glenoid and acromial arch. If such transplantations are to be carried out, the necessary depressor action and rotation of the head may be obtained by implantation of the latissimus dorsi and teres major to the postero-inferior aspect of the greater tubercle. Transfer of one of these muscles alone will not improve function. In one of our patients, for example, transplantation of the teres major to the greater tubercle gave no improvement in function except insofar as the postural activity of the muscle came into play, maintaining a static position once passively reached. The latissimus dorsi, being an internal rotator, inhibited motion when brought into play by voluntary assistance. On the other hand, had the latissimus dorsi been transplanted with the teres major, external rotation, fixation, and the necessary downward pull might have been anticipated.

In those cases in which the latissimus dorsi and teres major are paralyzed, fixation of the head of the humerus to the glenoid and acromion by the long head of the biceps, a modified Nicola procedure, will give some stability to the glenohumeral joint, and, with or without reinforcement of the deltoid, often results in better function, and is sometimes, particularly in women, to be preferred to arthrodesis of the joint.

If the pectoralis major is used to replace the anterior portion of the deltoid, it is essential to employ only its clavicular head, for this head is the sole portion of the muscle active in flexion of the extremity. The remaining sternocostal element comes into play with adduction and medial rotation, and, therefore, cannot be utilized because of its antagonistic action in this movement.

The following case is illustrative of the importance of fixation of the humeral head and the dual function of the pectoralis major:

E. W., female, thirteen years of age, entered the Children's Hospital on July 5, 1940.

History: The patient had had an attack of anterior poliomyelitis at the age of eight months, the residual paralysis being limited to the right shoulder and the right arm. At the time of her entry to the Hospital, the principal complaint was inability to abduct the arm.

Examination: There was extensive paralysis of the muscles of the shoulder, including the anterior half of the deltoid, the supraspinatus, infraspinatus, and teres minor muscles. Good power remained in the pectoralis major, the teres major, latissimus dorsi, trapezius, and serratus anterior. A forward subluxation of the head of the humerus occurred when an attempt was made to abduct the arm.

Operation: The operation was done in two stages. In the first stage, the scapulohumeral joint was stabilized by passing the long head of the biceps through the head of the humerus and then through the acromion process (a modified Nicola operation). The anterior half of the deltoid muscle was reinforced by transferring the origin of the clavicular head of the pectoralis major to the outer clavicle and acromion, with preservation of the blood and nerve supply of this muscle. The second stage of the operation consisted of a transplantation of the insertion of the clavicular head of the pectoralis major to the region where the deltoid is inserted upon the lateral aspect of the humerus.

The patient's convalescence was uneventful. The purpose of the operation was achieved, as subluxation of the head of the humerus was prevented and an active abduction of the arm to 170 degrees was obtained.

Although, after operation, the increased stability of the humeral head on the glenoid increased the efficiency of the posterior deltoid in abduction, and the patient was enabled to elevate actively the arm to 160 degrees, electromyographic studies showed that the transplanted clavicular head contracted only when the arm was raised in the plane of flexion. In abduction, the transplant was completely inert in spite of attempts at re-education. It is conceivable that transfer of the insertion of the sternocostal head of the pectoralis major to the postero-inferior aspect of the greater tubercle, thus converting it into a lateral rotator, would complement the action of the transplanted clavicular element. With voluntary contraction of the pectoralis, the muscle acts as a whole, and, even with re-education, it is impossible to contract any portion of the muscle independently. Therefore, transference of the sternocostal head to a position where it can act on voluntary contraction as a lateral rotator is essential. Otherwise, on re-education, this element will act as an adductor and medial rotator and oppose the transplant.

The recognition of the force-couple principle in the action of the muscles which move the scapula is also of value in planning treatment. The support of the scapula by means of the rhomboids, levator scapulae, and upper and middle segments of the trapezius enables the serratus anterior, together with the upper and lower portions of the trapezius, to act as components of the scapular force couple. In paralysis of the rhomboids or of the supporting portion of the trapezius, fascial transplant from the base of the scapular spine or its upper border to the lower cervical spinous processes (after the method of Lowman) may be of service in restoration of the fulcrum for action of this force couple.

The myographic studies of the basic activity in the living clearly reveal the nature of the pattern of muscular activity. There is, for example, no such thing as a prime mover, as ordinarily understood. There are only patterns of action. This conception extends and amplifies the axiom laid down by Beevor when he said with regard to the brain that it knows nothing of the action of the individual muscles, but only of movement. We can, then, see a central pattern of motion carried to the periphery. Furthermore, we are forced to recognize that the principles as adumbrated by MacKenzie are entirely incorrect. MacKenzie contends that an individual muscle has but one function. Sampling of various portions of an individual muscle shows that they can act independently, but synchronously in association with the total pattern of a specific movement. This great principle is absolutely fundamental for establishing rational procedures of muscle re-education. These patterns of muscular activity cannot be reduplicated by voluntary contraction of the muscle alone. They can only be brought into play by carrying out the precise motion itself, which probably brings into play proprioceptive mechanisms. It is our opinion that here lies the rational explanation for the empirical methods employed in the Kenny treatment of poliomyelitis. Motion, and motion alone, is the only known stimulus able to engender, in phase and degree, the muscle activity requisite for the establishment of the pattern as a whole. A further principle is demonstrated in the existence of certain muscles which play a static rôle in the maintenance of a posture of the extremity. These must be borne in mind when attempts are made to utilize such muscles as kinetic elements in reconstruction procedures. This static action has been confirmed clinically, for example, when the teres major, transplanted to replace the infraspinatus group, failed to act during motion of the extremity and came into play forcibly only when a static position was maintained.

REFERENCES

BEEVOR, C. E.: The Croonian Lectures on Muscular Movements and Their Representation in the Central Nervous System. London, Adlard & Son, 1904.

CALDWELL, G. D.: Treatment of Complete Permanent Acromioclavicular Dislocation by Surgical Arthrodesis. J. Bone and Joint Surg., XXV, 368, Apr. 1943.

CODMAN, E. A.: The Shoulder. Rupture of the Supraspinatus Tendon and Other Lesions in or about the Subacromial Bursa. Privately printed Boston, 1934.

MACKENZIE, W. C.: The Action of Muscles, Including Muscle Rest and Muscle Re-Education. New York, 1921.

LESIONS OF THE MUSCULOTENDINOUS CUFF OF THE SHOULDER *

I. The Exposure and Treatment of Tears with Retraction

BY HARRISON L. McLAUGHLIN, M.D., NEW YORK, N. Y.

From the Fracture Service of the Presbyterian Hospital in New York, and the Department of Surgery, the College of Physicians and Surgeons, Columbia University

Dr. E. A. Codman repeatedly pointed out the importance of the subacromial structures in connection with the subject of "painful shoulder". He mentioned the great prevalence of this complaint, and emphasized the tremendous total of man hours lost to industry because of it. Under the stimulus of his teachings, a considerable number of these cases have been studied by the Fracture Service of the Presbyterian Hospital in New York.

It is now fashionable, although incorrect, to label most sore shoulders devoid of fracture as "bursitis". Seldom is it realized that the term bursitis is no more a diagnosis than headache or jaundice, and that, when present, it usually constitutes a second condition having no connection with the primary lesion other than proximity. It is the purpose of this paper to discuss the exposure and therapy for one of these primary conditions,—namely, a tear of the intrinsic musculotendinous cuff with retraction.

The commonly used approaches to the shoulder-joint region almost invariably are accompanied by one or more of the following handicaps:

1. Inadequate exposure due to obstruction by an intact acromion[5].
2. A poor cosmetic result due to spreading of a vertical scar.
3. Paralysis and wasting in the anterior portion of the deltoid, due to operative interference with axillary nerve branches[14].

Evaluation of the merits and disadvantages of the available routes has resulted in the use of a superior approach, primarily based on the saber-cut incision advocated by Codman, and the posterior approach to the shoulder described by Kocher. This approach not only eliminates risks Nos. 2 and 3, but also affords an excellent survey of the whole joint region, including the inside of the articular cavity, and, in addition, presents certain distinct intrinsic advantages affecting postoperative care and rehabilitation.

The skin incision parallels the strap or suspender line, and the normal skin creases of the region; it is situated just lateral to the acromioclavicular joint, and extends from the posterior to a point three to five centimeters in front of the anterior border of the acromion (Figs. 1-A and 1-B). That portion anterior to the acromion is deepened, and the underlying deltoid fibers are separated and retracted to expose the roof of the bursa and the coraco-acromial ligament, which are in turn incised and retracted. An exploratory opening (Figs. 2-A and 2-B) is then available through which, by manipulation of the extremity, almost all except the most posterior and inferior portions of the bursa and musculotendinous cuff may be examined, the pathology evaluated, and minor procedures, such as the excision of the bursa or evacuation of calcified deposits, carried out.

At this stage of the approach it should be emphasized that:

1. All deltoid splitting should be near enough to the anterior border of the muscle so that, if terminal axillary nerve fibers are accidentally severed, the amount of resultant atrophy and paralysis will be insignificant, both from a cosmetic and a functional viewpoint.
2. Deltoid splitting should be carried down from, rather than up to, the anterior border of the acromion. In this way no unnecessary splitting is done; danger of involving axillary nerve branches by dissection is minimized; and the circumflex acromial vessels may be identified and controlled at the start, ensuring a dry field.

* Read at the Annual Meeting of The American Orthopaedic Association, Cleveland, Ohio, June 8, 1943.

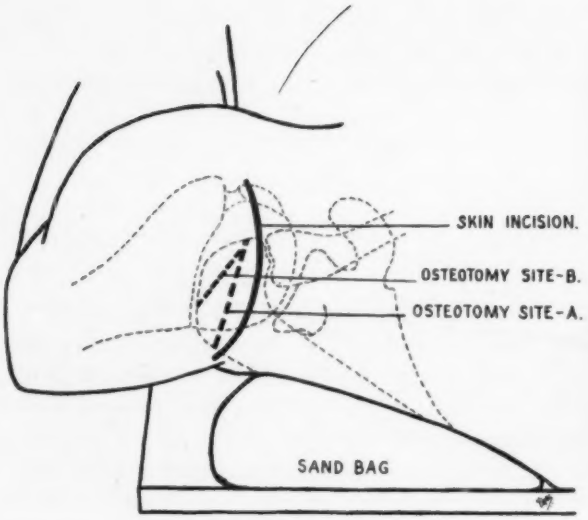


FIG. 1-A

FIG. 1-B

The left shoulder viewed from above, showing the skin incision and the sites for division of the acromion.

3. When more complete exposure of the anterior joint region is desired, separation of the deltoid fibers may readily be extended, but each additional centimeter of extension increases the risk of damage to the axillary nerve branches. It is safe to split the proximal four centimeters of the anterior deltoid, and more than this is required only in the exceptional case.

When examination through this exploratory opening has indicated the need for a complete exposure of the joint region, the skin incision is carried directly backward to the posterior border of the acromion and deepened to bone. The acromion is then divided by an osteotome (Figs. 3-A and 3-B). When intra-articular exploration involving complete exposure of the glenoid is required, the osteotomy is directly anteroposterior and about midway between the acromioclavicular joint and the lateral border of the acromion (Fig. 1-A, *Site A*); but, for purposes of repair work on the intrinsic muscle cuff, a more oblique osteotomy (Fig. 1-A, *Site B*), deviating laterally to emerge at about the lateral tip of the acromion, gives adequate exposure and a little better cosmetic result. The loose areolar tissue is next separated from the undersurface of the divided fragment, following which the

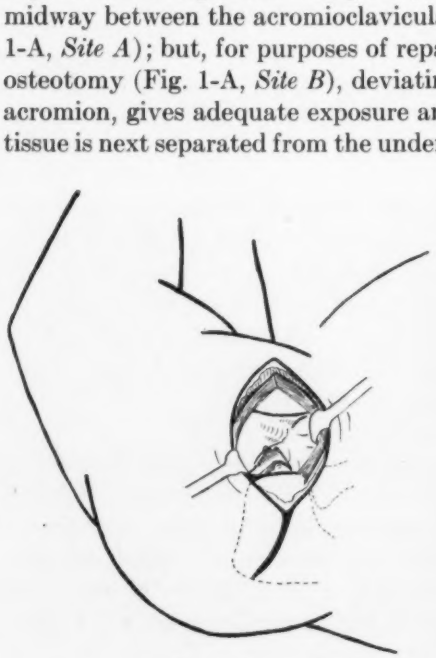


FIG. 2-A

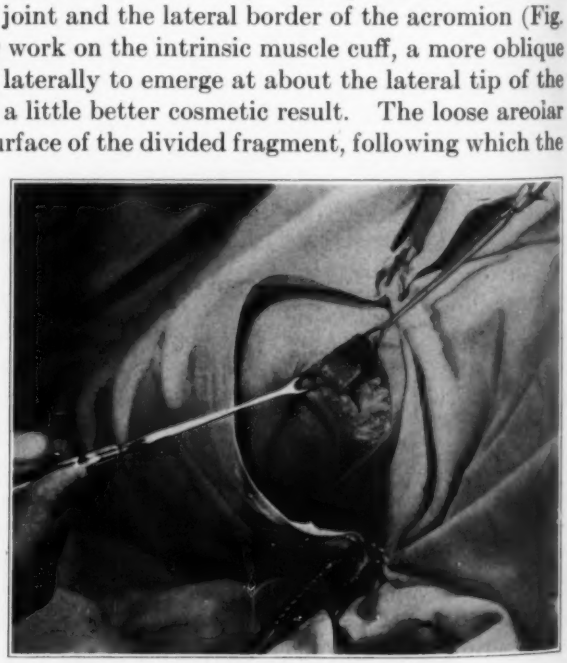


FIG. 2-B

The left shoulder viewed from above, showing the exploratory portion of the incision.

FIG. 3-A

FIG. 3-B

Viewed from above, showing the division of the acromion.

outer flap of the wound is reflected laterally as far as the level of the surgical neck of the humerus, making available for inspection the humeral head and the tuberosities covered by the intrinsic muscle cuff (Figs. 4-A and 4-B).

A complete survey of the interior of the joint (Figs. 5-A and 5-B), is obtained by making a longitudinal incision through the cuff from the level of the glenoid cavity to the anatomical neck of the humerus. This incision parallels the direction of the cuff fibers, and may be made through any one of the four tendons, but is most useful and least traumatic when located in the aponeurosis joining the subscapularis and supraspinatus tendons.

Through such an opening in the cuff, it is found possible to do any of the common shoulder-joint procedures with ease. A dislocated fragment of the humeral head may be removed from above, rather than through the brachial plexus. Reattachment of the labrum glenoidale or anterior capsule for recurrent dislocation[1], is carried out from within the joint, thus avoiding the trauma caused by transverse section of the coracoid and subscapularis. Transplantation of the biceps tendon[16] is done with the added certainty of knowing exactly where the tendon is to emerge from the humeral head, since the bony tunnel is made from above downward, and the intra-articular point of emergence of the tendon

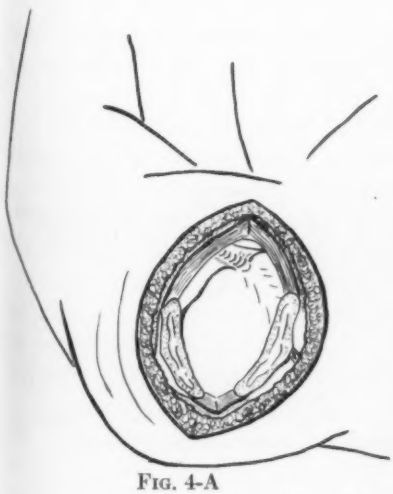


FIG. 4-A

FIG. 4-B

Viewed from above, showing the exposure of the musculotendinous cuff.

may be placed as far medially as desired. Reassemblement and fixation of complicated fractures and fracture-dislocations, or reconstruction procedures, such as that described by Jones[10], are much more simply done, since the whole pathological field is in full view at one time and the soft-part tears invariably accompanying such lesions are amenable to easy repair.

Multiple sutures of fairly fine silk are used for side-to-side closure of the longitudinal incision in the cuff, and it can be demonstrated by passive manipulation of the extremity at operation that the tension on such a repair is insignificant. Consequently, the fact that the intrinsic muscle cuff has been

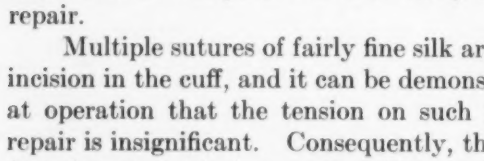


FIG. 5-A

FIG. 5-B

Viewed from above, showing the exposure possible through a longitudinal incision in the cuff.

opened does not constitute a factor tending to delay mobilization, nor has it been observed to delay convalescence to any appreciable extent. It should be emphasized, however, that such repair must be secure enough to prevent early mobilization from pumping joint fluid through the suture line to collect in the region of the bursa and exert a deleterious effect on tissue healing.

In closing the wound proper, it is considered advisable to excise and discard the outer fragment of the acromion. The stump is beveled off and reshaped for purposes of obtaining the optimum cosmetic result, and the free deltoid origin is sutured to the rather tough tissue on its superior aspect. If for any reason the fragment is not removed, wound closure is accomplished by soft-tissue suture only; no attempt at bone fixation should be made. Good union of the bony fragment has occurred in all such cases whether or not fixation was present.

Removal of this fragment has been followed by no functional defects. On the contrary, it has appeared to be of distinct benefit in most cases. Smith-Petersen has pointed out that removal of the lateral portion of the acromion in itself does no harm, and is successful in relieving certain types of shoulder pain. The experience gained in observing the postoperative course of shoulder cases, in which removal has been done in addition to other procedures, has verified his statements. There has been noted not only a distinct decrease in the amount of postoperative pain suffered by these patients, but also a distinct increase in the ease with which they are able to commence active mobilization of the extremity, as compared with previous cases in which no division of the acromion was done, or in which division was followed by fixation of the fragments.

Further potential benefits are gained by thus reducing the size of the acromion. If a ruptured cuff has been repaired, such a reduction should mean less surface against which the repair site will impinge on elevation of the arm,— consequently less wear, less postoper-

ative pain, and earlier rehabilitation. If operation for recurrent dislocation has been done, a smaller acromion should mean a less prominent fulcrum over which the humerus may subsequently be levered towards a dislocated position, and a decrease in the potential risk of recurrence. In cases of fracture-dislocation requiring removal of the humeral head, shortening of the acromion and the resultant transplantation of the corresponding deltoid origin to a point more medial than normal should result in a greater mechanical advantage for the deltoid and greater power of abduction. All of these premises now have been adequately confirmed by clinical experience and will be reported in detail at a later date.

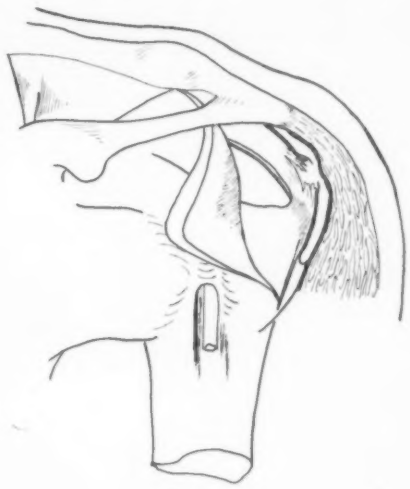


FIG. 6

Left shoulder viewed from in front, showing an incomplete tear involving the surface of the cuff.

The wound, *per se*, requires no particular aftercare, and the postoperative management of the extremity may be dependent upon the condition for which the operation was done. Immediate mobilization has not resulted in any tendency for the deltoid to separate from its transplanted position, but it is considered advisable to have the patient refrain from active elevation or abduction against gravity for a period of about two weeks, after which active raising of the arm with support may be commenced.

More than 3,000 painful shoulders have been treated in the Fracture Service of the Presbyterian Hospital in New York in the past ten years, and the statements to be made in this and subsequent reports are based upon clinical and follow-up observations made on this group of patients. Two hundred and seventy-five operations performed for lesions of the musculotendinous cuff and its associated structures have demonstrated that no two lesions are ever alike and no two lesions ever call for identical operative technique. The diversity of the pathological picture in the case of the ruptures is especially striking, but enough have now been studied at operation to permit of a tentative classification [2] according to pathological anatomy and the therapy required. Such a classification relates only to rupture of the tendinous portion of the cuff, and, for the most part, excludes the various

FIG. 7

Viewed from in front, showing an incomplete tear involving neither surface of the cuff.

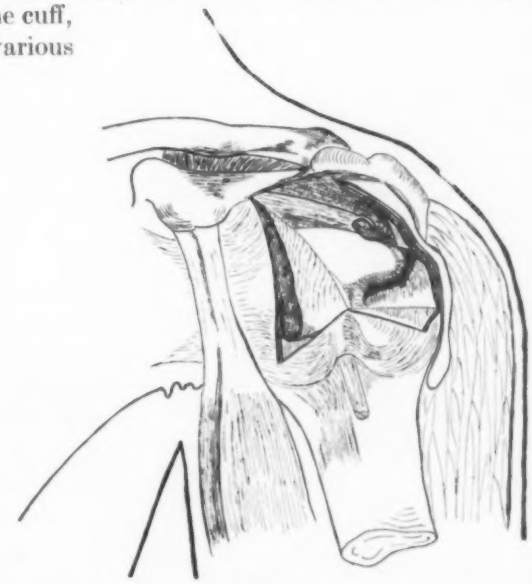


FIG. 8

Viewed from in front, showing an incomplete tear involving the deep surface of the cuff.

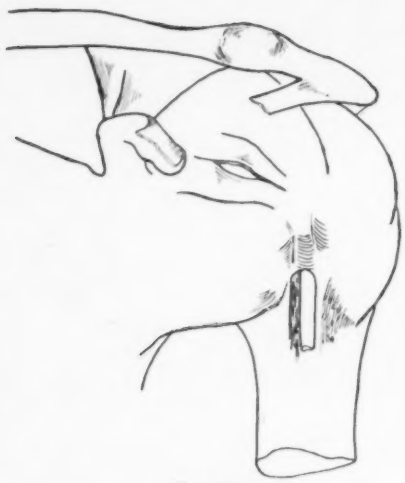


FIG. 9
Viewed from in front, showing a small longitudinal split.

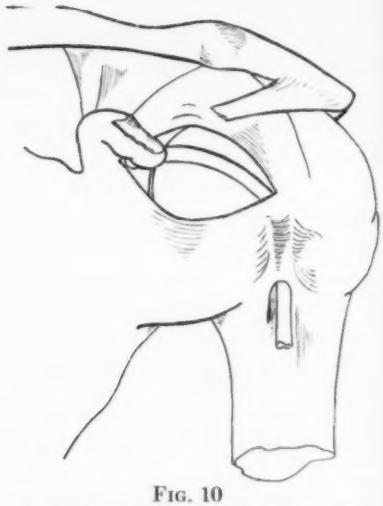


FIG. 10
Viewed from in front, showing a large longitudinal split.

capsular and ligamentous lesions accompanying dislocations and other more violent injuries, which will be made the subject of a subsequent report.

I. INCOMPLETE RUPTURE

Such a tear is incomplete in the sense that it does not involve the whole thickness of the cuff. It may be situated on the superficial surface of the cuff, so that the resultant irregularity in the floor of the bursa produces a constant source of chronic irritation on motion of the joint (Fig. 6). It may be located within the substance of the cuff, so that it involves neither surface (Fig. 7); and there is evidence to indicate that the deposition of calcium salts in the tendons constitutes one manifestation of the poor healing qualities of such a lesion. On the other hand, it commonly involves only the deep surface of the cuff, so that the retracted tab of tissue may produce an internal derangement of the shoulder joint in much the same way that a mobile tab of medial meniscus produces symptoms in the knee (Fig. 8).

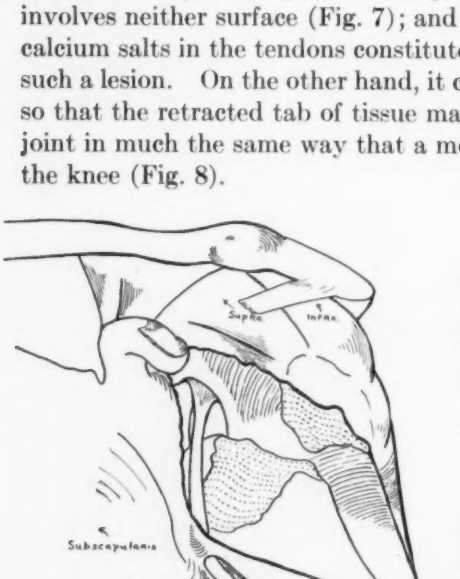


FIG. 11
Viewed from in front, showing the mechanics of a dislocation accompanied by fracture of the tuberosity.

II. COMPLETE RUPTURE

Such a tear is complete in the sense that it involves the whole thickness of the cuff and results in a direct communication between the subdeltoid bursa and the joint cavity. This group of lesions may be divided into four main categories.

A. *Pure Transverse Rupture*

This is an uncommon lesion. It has not been encountered in a young person with a healthy cuff, and is usually to be found in the age group past thirty-five, and in persons whose cuff has become weakened by attrition.[12,15] Because it is purely transverse, with both ends of the torn portion adherent to the adjoining intact cuff, minimal retraction is the rule; and for this reason such tears are amenable to direct end-to-end suture or reinsertion into the anatomical neck of the humerus as described by Wilson, without undue tension on the repair site.

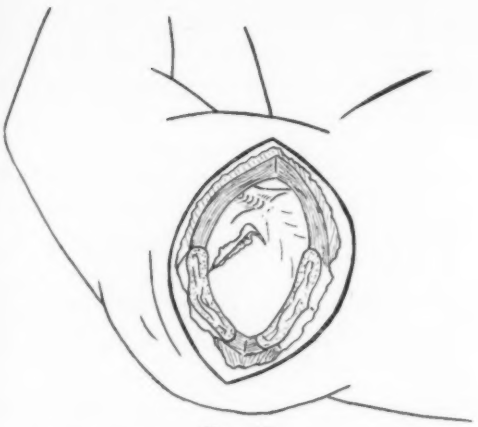


FIG. 12
Viewed from above, illustrating the beginning of a longitudinal split superimposed on a primary transverse tear.

FIG. 13
Viewed from above, illustrating the commencement of retraction following extension of the split.

B. *Pure Vertical Rents or Longitudinal Splits Paralleling the Direction of the Cuff Fibers*

Such a tear is most frequently found in young persons, whose tendinous cuff is thick and strong enough to withstand transverse rupture, and invariably has been situated in the aponeurosis uniting the subscapularis and supraspinatus,—that portion of the cuff aptly described by Jones as the coracocapsular ligament.[11] A small split of this type (Fig. 9), probably heals spontaneously under favorable circumstances, but on occasion has been found to give rise to protracted symptoms requiring late repair. In a longer split, however, the edges are pulled apart by the divergent forces of the subscapularis and the external rotators, and an elliptical hiatus is produced in the anterosuperior part of the cuff (Fig. 10). The largest such lesion—three by two inches—found to date was in a teen-aged girl, and was accompanied by a gross subluxation of the humeral head which disappeared following side-to-side repair of the ununited rent.

Commonly, the splitting apart of the fibers extends as far laterally as the level of the surgical neck of the humerus, and, as it passes over the greater tuberosity, involves the bone (in the form of a fracture of the tuberosity) as well as the overlying tendinous envelope. Under such circumstances, the humeral head loses the support of its posterior and superior stabilizing muscles, and, aided by the unopposed pull of the intact subscapularis, rolls downward and forward into a dislocated position (Fig. 11). Such dislocations are found to be intracapsular in nature, with the anteroinferior joint structures essentially intact.[8,18]

The various types of cuff pathology so far mentioned contain material worthy of more detailed discussion, and this subject will be taken up at greater length in subsequent papers. They have been described briefly as a prelude to facilitate comprehension of the traumatic and posttraumatic mechanics involved, and the rationale underlying the therapy advised for the following most common of all the cuff ruptures.

C. *The Tear with Retraction*

The general conception of a retracted tear of the cuff has been of a more or less transverse

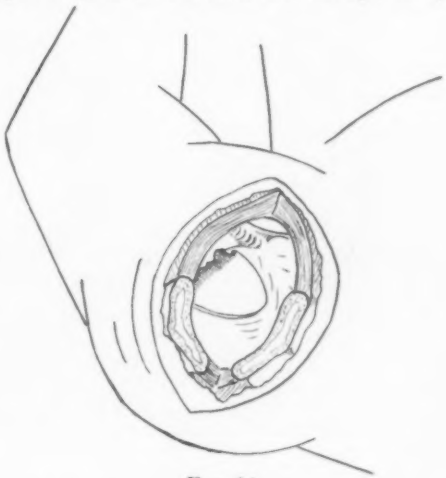


FIG. 14
Viewed from above, showing the eventual form of the tear following retraction.

rupture extending across the fibers or avulsing the tendon from its bony insertion, and the suggested methods of repair[2,3,4,5,6,7,9,18,21], have been designed to combat this type of lesion by end-to-end suture or reinsertion of the retracted fragment into the anatomical neck of the humerus. It is probably safe to say that this is a misconception, which has been, and is, the most important single factor in the production of unsatisfactory results, because a transverse rupture is seldom found alone, and pure end-to-end repair is frequently contra-indicated. Before a rational plan of repair can be conceived, an understanding of the following characteristics of the mechanics of the rupture is necessary.

In the first place, such ruptures seldom occur except in tendinous cuffs that have been subjected to sufficient use and attrition to make them thin and weak, and the seat of scarring and degenerative changes.[12,15] This fact must be taken into consideration, and the degenerated scarred peripheral edges of any tear must be excised back to normal vascular tissue before repair may be done with optimum chances for healing.[4,21]

In the second place, while immediate operations on fresh injuries have indicated that the primary tear is usually transverse in nature, explorations at various later periods following injury have demonstrated that certain changes in the nature of the lesion quickly and inevitably occur. The cuff might be likened to a sheet of cellophane which, so long as it remains intact has great tensile strength, but which, once a small rip is started, progressively tears more and more with each additional strain placed upon it. The corresponding lesion taking place in the tendinous cuff consists of a progressive splitting apart of the fibers, commencing at one end of the original transverse rupture (usually the anterior end) and extending medially to produce a longitudinal split parallel to the long axis of the fibers (Fig. 12).

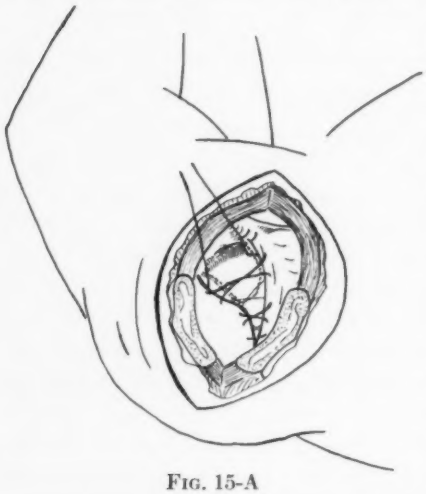


FIG. 15-A

Undoubtedly some injuries are such that both the transverse and splitting portions of the tear are produced simultaneously, but more important is the fact that, once established, the extent of both portions may be increased by minor traumata or sudden

FIG. 15-B

FIG. 15-C

Figs. 15-A, 15-B, and 15-C: Viewed from above, showing the cuff excision, and placement and action of the continuous suture.

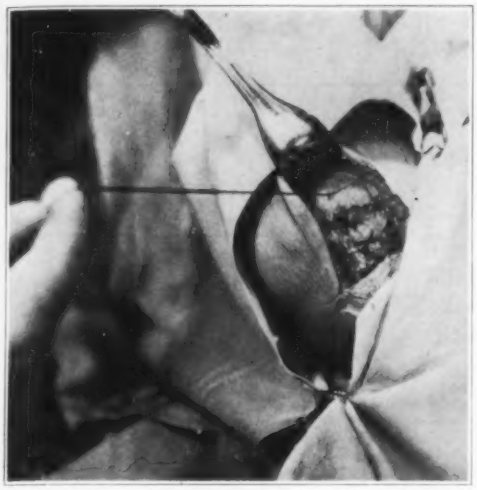


FIG. 16-A

FIG. 16-B

Viewed from above, showing the continuous suture tied lateral to the tuberosity.

movements of the extremity. At operations on patients whose acute symptoms dated from a recent injury, it has been noted repeatedly that the cuff pathology consisted of a rupture of obviously long duration upon which was superimposed a recent extension of the pre-existing tear.

As the splitting portion of the lesion extends medially, it becomes subject to a new mechanical force. The subscapularis pulls its anterior edge forward, while the external rotators pull its posterior edge backward; and in this manner the typical crescentic or triangular hiatus begins to form (Fig. 13). Time and cicatrization further camouflage the true nature of the lesion, so that on superficial examination it appears to be nothing more than retraction superimposed on a transverse tear (Fig. 14). Attempts to repair this type of lesion by end-to-end suture or by reinsertion of what appeared to be the retracted end of a transverse tear into the anatomical neck of the humerus, especially if the gap to be closed was further increased by excision of the scarred avascular periphery of the tear, too often have been unhappy experiences both for the patient and the surgeon.

FIG. 16-C

Showing the additional mattress sutures frequently required.

As a result of the realization that a retracted lesion must be a combination of the transverse and longitudinal tear, a corresponding change in the method of repair has been developed. Actually the change is in principle rather than method, and is characterized by two important departures from the generally used technique of end-to-end repair. The first is that the size of the hiatus in the cuff and, *ipso facto*, the amount of retraction, is decreased by side-to-side rather than by end-to-end repair. Codman recognized that such a procedure would be much easier than the end-to-end method, but considered it unwarranted, because it appeared to distort the normal anatomical mechanics of the structures involved. The second is that no attempt is made to restore normal anatomical reposition of the structures involved past the point of tension. Reinsertion of the retracted portion of cuff into the humeral head is done at whatever point it will reach with ease, with the arm

TABLE I

MASSIVE AVULSION OF CUFF INSERTION (8 CASES)

Case	Preoperative Disability Time	Degree of Avulsion	Hospitalized Postoperatively (*Days*)	Work	Resumed Light Work	Resumed Regular Work	Present Symptoms	Present Motion Defect	Present Functional Defect	Present Postoperative Period
1. A. K.	30 days.	All of cuff avulsed except part of teres and subscapularis.	14	Carpenter.	2 months.	2½ months.	None.	10 to 15 degrees of defect in internal rotation.	None.	3½ years.
2. P. G.[1]	3 months.	All external rotators and part of subscapularis avulsed.	16	Riding-master, groom.	2 months.	3 months.	Occasional weather ache.[2]	None.	None.	3½ years.
3. C. F.	1 month.	Complete avulsion. Cuff lay between humerus and glenoid.	11	Superintendent.	6 weeks.	2 months.	None.	Elevation to 150 degrees.	Difficulty washing high windows only.	3 years.
4. J. G.	3 weeks.	Complete avulsion of supraspinatus and infraspinatus, and partial avulsion of subscapularis.	12	Helper on moving van.	1 month.	2 months.	None.	Elevation to 165 degrees.	None.	1 year.
5. O. R.	8 months.	Complete avulsion of cuff except for a portion of subscapularis.	12	Machinist and carpenter.	6 weeks.	4 months.	None.[3]	None.	Weak for heavy lifting.	2½ years.
6. S. K.	1 week.	Complete avulsion of external rotators. Subscapularis intact.	12	Helper on moving van.	5 weeks.[4]	6 months.	None.	Elevation to 170 degrees.	Slightly weak for heavy lifting.	2 years.
7. G. H.[1]	5 months.	Complete avulsion of external rotators. Subscapularis intact.	13	Riveter.			Pain, weakness, and tenderness.	Marked defect in all planes.	Claims complete disability.	1 year.
8. L. L.	6 months.	Complete avulsion of all cuff attachments.	Too recently operated upon for analysis.							

[1] Case is discussed under "Postoperative Care".
[2] The wire sutures were removed two years postoperatively.
[3] Patient reports by letter, but has had no recent examination.
[4] A micro-aerophilic infection developed six weeks postoperatively.

FIG. 17

Viewed from the side, illustrating a massive avulsion.

at the side, rather than at its normal point of insertion, the anatomical neck. No specific technique can be laid down for the repair of all retracted lesions, since their diversity of shape and size demands different methods; some require mainly a side-to-side repair while in others the main part of the repair will be end-to-end. Some combination of the two is usually indicated.

One prerequisite of any efficient repair is good exposure and adequate working room. The transacromial approach, utilizing the oblique type of osteotomy, has been found superior to any other for this purpose. With the tear completely exposed, its degenerated and scarred edges are excised back to viable tissue. The crescentic hiatus is then made into a long V-shaped opening which points medially (Fig. 15-A).

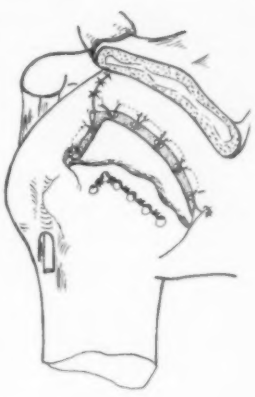


FIG. 18

Viewed from the side, showing method of repair for massive avulsion.

A shoe-lace type of continuous suture is started at the medial extremity of this opening, and is placed so that traction on its ends pulls the two edges of the opening together side-by-side (Fig. 15-C). This suture is pulled snug up to, but not past the point of tension. When this has been accomplished, it will be found that only a small V-shaped opening remains. The area of the humeral head visible through this residual opening is next denuded of articular cartilage, so that the edges of the opening come into contact with raw bone, and the two ends of the continuous suture are passed through drill holes and are tied at the outer surface of the greater tuberosity (Fig. 16-A). Frequently the residual gap in the cuff is so large that the stabilization produced by the main suture is not sufficient to anchor its edges down securely to the denuded area of the humeral head. When this is the case, additional mattress sutures are required (Fig. 16-C).

The net result of such a procedure is that two healthy vascular cuff edges are brought together, and are held with minimal tension by the main continuous suture. Inasmuch as the suture is tied on the lateral surface of the bone, any additional tension subsequently placed upon it by movement of the extremity will tend to pull these edges together rather than apart. The remaining lips of the cuff opening are fixed in apposition to raw bone surface in whatever location they will remain without tension, with the arm at the side. At times small ruptures with mild retraction may be excised and fixed directly to the bone underlying the edges of the tear, but such cases seem prone to a more uncomfortable and protracted rehabilitation period than similar cases in which a side-to-side revision has been done. The technique described has the theoretical disadvantage of some small distortion of the mechanics of the joint by failure to secure normal anatomical reposition of the tendons involved. However, the advantages of a snug but tension-free repair—no matter what the shape of the tear or the amount of retraction—of not having to maintain the extremity in an abducted position for fear of disruption, and of early active motion and use of the arm are practical and important.

D. *Massive Avulsion of the Cuff*

The mildness of the defect resulting from even gross transplantation of the cuff insertion is well illustrated by the results in the eight cases, shown in Table I, which belong in a class by themselves. All were similar to what Bosworth[3], has called a massive avulsion of the cuff,—that is, a complete avulsion of the external rotators, with retraction almost to the level of the glenoid made possible by a longitudinal split completely separating the supra-

[3] Patient reports by letter, but has had no recent examination.
[4] A micro-aerophilic infection developed six weeks postoperatively.
[5] The wire sutures were removed two years postoperatively.

spinatus from the subscapularis (Fig. 17), and at times involving the tendon of the latter as well. At times the retracted cuff becomes interposed between the glenoid and humerus. Symptoms of pressure on the brachial plexus, resulting from the almost invariable chronic subluxation of the humerus, are common. All the lesions were repaired in a manner similar to that depicted in Figure 18,—by as much side-to-side suture as could be managed without tension, and by reinsertion of the cuff into the superior surface of the humeral head, so that its suspensory function, as described by Jones, was maintained. The most recent patient, who is still in the early postoperative stage and whose result cannot as yet be evaluated, had a Nicola transplantation of the long head of the biceps done in addition to the cuff repair. It is possible that this should be done in all such cases, but none of the other seven patients has shown any tendency toward subluxation of the humeral head when the lesion was repaired by reinsertion of the cuff alone. Therefore, it is considered that the addition of a Nicola procedure does not give more than a little extra assurance of stability of the humeral head, and is not required except in the occasional case where the reinserted cuff appears to give inadequate stability and suspension as a result of its grossly transplanted insertion.

Three of the patients in this group have been followed more than three years, two of them more than two years, and two of them for a year. One patient, a fifty-year-old foreign laborer and a compensation case, was a dismal failure. An inadequate repair was done, requiring the use of a postoperative abduction spica which had to be removed at an early date because of claustrophobia. Undoubtedly the repair disrupted, and one year later he was still unable to elevate the arm to the horizontal, and complained of much pain and complete disability for heavy work. The other six patients were all back at light work within two months, and four of them were able to do regular heavy work within three months. The fifth was back at manual labor in four months, and in the sixth patient a micro-aerophilic infection developed in the wound six weeks following operation; as a result he did not return to his regular work as a helper on a moving van until the sixth month. All except one were subjected to relatively gross transplantation of the cuff insertion; yet three of them enjoy unlimited elevation of the extremity, and the other three elevate to 150, 165, and 170 degrees, respectively. One, a seventy-year-old riding master, complains of an occasional mild ache; and another, a sixty-year-old carpenter, complains that his arm is not so strong as it used to be, but both have unlimited motion. None of the other patients have any residual symptoms.

ANALYSIS OF RESULTS

Any statistical analysis without a detailed discussion of the many individual factors involved is of doubtful value. More than seventy-five cuff ruptures of various types have been repaired, but a table of the total results, which would include accompanying lesions or other local complications, as well as cases marked by symptoms without disability, would mean little, and would present a confusing picture. The results in seven massive avulsions have been mentioned, and, for the purposes of this paper, further evaluation will be restricted to a group of thirty-two cases. Each one satisfies the following criteria, which constitute an adequate common denominator upon which the results can be estimated, and from which certain broad principles relative to the management of cuff tears may be drawn. Each patient in this series:

1. Did manual work or its equivalent;
2. Was unable to do regular work prior to operation;
3. Had repair of a retracted cuff tear uncomplicated by any other local pathology;
4. Has been followed for at least six months.

Analysis of Disability Time

None of these patients suffer from any economic or functional disability. Twenty-

two were known to be back at their former manual jobs or equivalent work within two months, while in three others the records are not sufficiently explicit about time and type of work to permit of their inclusion. The convalescence of the remaining seven was delayed in each case by one extraneous circumstance apart from the local condition.

Case 31 (Table II), T. P., was complicated by a postoperative infection that fortunately did not involve the site of repair, but did hold up the patient's return to normal strenuous activities for a period of six months. He is now without limitation of motion or symptoms and carries out the duties of a paratrooper with ease.

Case 20 (Table II), A. M., was complicated by a furuncle which was not related to the operative incision, but which served to delay the patient's return to work until three months after operation.

Case 2 (Table II), N. B. The patient was a plasterer by trade and while he had no difficulty working on side walls, he was unable to work without trouble on ceilings for almost nine months. At that time he had no symptoms or limitation of motion.

Case 22 (Table II), L. S. The patient was a Mexican who spoke practically no English, and who was mentally unequipped to cooperate in a program of rehabilitation exercises. In spite of this, she returned to full work as a general domestic servant three months following operation. Three years later she showed a keloid scar, but no motion defect, and she complained of no symptoms.

Three other patients were under Workmen's Compensation, and will be reported hereafter under that specific category.

Residual Motion Defect

Twenty-two patients showed no limitation of motion at the time of their last follow-up examination, and in one case the record was considered inadequate for analysis. Five patients showed a small defect in rotation. While these defects were objectively measureable, none of them were subjectively significant to the patient. Only two patients showed a limitation of elevation, −20 and 10 degrees, respectively. Two others had a small motion defect in all planes.

Residual Symptoms

Twenty-one patients were completely asymptomatic when last examined. Nine had, as their only complaint, an occasional ache aggravated by changes in weather. One complained of a grating sensation on motion of the arm, and in another the notes were again inadequate for analysis.

The Duration of the Period Prior to Repair

The symptoms and disability prior to operation varied from one day to several years. This is of interest in view of the uniform postoperative behavior of the great majority of the cases, because it has been stated repeatedly by Codman, Bosworth, and others that the results in these cases should vary directly as the speed with which they are repaired following injury. As a result of the evidence presented by this series, it is felt that this aspect of the problem has been overemphasized, and that, in general, regardless of how soon operation is done, the lesion is already of long standing in the sense that degenerative and attritional changes in the cuff substance have preceded the actual rupture, probably by a matter of years[15]. There does not seem to be any doubt that the earlier the operation is done, the smaller the tear will be, since most ruptures tend to increase in size progressively up to a certain extent. On the other hand, many such lesions never give symptoms and disability enough to warrant operation, and, for this reason, a waiting period of at least two weeks[3], during which time the extremity is kept protected from further trauma and under a regimen of conservative therapy, is considered to be the course of wisdom in acute injuries suggestive of cuff tear.

TABLE II

UNCOMPLICATED RETRACTED TEARS OF THE MUSCULOTENDINOUS CUFF OF THE SHOULDER (32 CASES)

Case	Preoperative Disability Time	Degree of Retraction (*Centimeters*)	Hospitalized Postoperatively (*Days*)	Work	Resumed Light Work	Resumed Regular Work	Present Symptoms	Present Motion Defect	Present Functional Defect	Present Postoperative Period
1. L. M.	2 weeks.	2 to 3	8	Laborer.	6 weeks.	Uncertain.	None.	Questionable defect in elevation.	None.	5 years.
2. N. B.[1]	1 month.	3	15	Plasterer.	6 weeks.	9 months.	None.	None.	None.	5 years.
3. C. F.	2½ years.	2 to 3	14	Athlete.	3 weeks.	8 weeks.	Occasional weather ache.	None.	None.	1 year.
4. M. M.	3 weeks.	2	16	Housewife, does all work.	Unrecorded.	2 months.	Occasional weather ache.	None.	None.	3 years.
5. H. H.	6 months.	1.5	7	Carpenter.	Unrecorded.	2 months.				
6. L. W.	1 year.	1	8	Athlete.	1 month.	6 weeks.	None.	None.	None.	1 year.
7. T. S.	2 weeks.	2	9	Laborer.	2 months.	2 months.	None.[2]	None.[2]	None.[2]	6 months.
8. M. A.	4 months.	2	13	Machinist.	1 month.	7 weeks.	Occasional weather ache.	None.	None.	6 months.
9. M. G.	9 months.	1	9	Domestic.	1 month.	1 month.	Occasional weather ache.	None.	None.	6 months.
10. B. G.	More than 1 month.	1	4	Housewife, tennis, golf.	1 week.	3 weeks.	Occasional weather ache.	None.	None.	6 months.
11. J. F.[3]	3 months.	2.5	12	Furnace stoker.	6 weeks.	2 months.	None.	None.	None.	6 months.
12. N. B.	10 months.	2 to 3	11	Housewife, does all work.	Unrecorded.	Uncertain.	None.	None.	None.	3½ years.
13. V. S.	4 weeks.	2	18	Domestic.	1 month.	2 months.	None.	None.	None.	3 years.
14. J. S.[3]	Many years.	2	21	Laborer.	2 months.	2 months.	None.[5]	None.	None.	2½ years.

13. V. S.	4 weeks.	2	18	Domestic.	1 month.	2 months.	None.	None.	None.	3 years.
14. J. S.[2]	Many years.	2	21	Laborer.	2 months.	2 months.	None.[5]	None.	None.	2½ years.
15. C. M.	10 weeks.	2 plus	20	Carpenter.	Unrecorded.	2 months.	Occasional mild pain.	Small defect in internal rotation.	None.	3 years.
16. G. R.	30 days.	2	7	Housewife, does all work.	1 week.	2 weeks.	None.[4]	Small defect in internal rotation.	None.	3½ years.
17. F. P.[3]	1 day.	1	4	Laborer.	2 weeks.	4 weeks (able), 2 months (actual).	None.	Small defect in internal rotation.	None.	3½ years.
18. L. M.	1 month.	2	6		6 weeks.	8 weeks.	Occasional mild ache.	Small defect in internal rotation.	Unknown.	6 months.[6]
19. J. F.[3]	1 month.	2 to 3	14	Carpenter.	4 months ?	18 months.	None.	Small defect in internal rotation.	None.	3½ years.
20. A. M.[1]	1 week.	2 to 3	14	Building superintendent.	5 weeks.	3 months.	None.	Small defect in internal rotation.	None.	3½ years.
21. A. M.	5 weeks.	3	21	Superintendent.	Unrecorded.	2 months.	None.	Small defect in internal rotation.	None.	3 years.
22. L. S.[1]	4 weeks.	1	11	Domestic.	Unrecorded.	3 months.	Occasional ache.	Small defect in internal rotation.	None.	3 years.
23. L. G.[7]	2 months.	2.5	17	Housewife, does all work.	Unrecorded.	Unrecorded.	None.	20-degree defect in elevation at 6 months.	Unrecorded.	6 months.

TABLE II (*Continued*)

Case	Preoperative Disability Time	Degree of Retraction (*Centimeters*)	Hospitalized Postoperatively (*Days*)	Work	Resumed Light Work	Resumed Regular Work	Present Symptoms	Present Motion Defect	Present Functional Defect	Present Postoperative Period
24. M. M.[3]	1 year.	2 to 3	14	Carpenter.	Many months.		None.	Unknown.[2]		2 years.
25. E. W.	8 months.	1	11	Domestic.	5 weeks.	7 weeks.	None.	Slight defect, all planes.	None.	9 months.
26. E. B.	1 day.	2	10	Housewife, does all work.	2 weeks.	3 weeks.	None.	Small defect in rotation.	None.	1½ years.
27. G. C.	4 months.	1 to 2	12	Laborer.	Unrecorded.	2 months.	Sense of grating on motion.	Small defect in internal rotation.	None.	1¾ years.
28. A. D.	3 months.	3	12	Mechanic.	Unrecorded.	Uncertain.	None.	20-degree defect in external rotation.	None.	6 months.
29. T. B.[3]	3 weeks.	1 to 2	18	Mechanic.	1 month.	6 weeks.	Occasional bad weather ache.	Slight defect, all planes.	None.	1 year.
30. A. B.	2 weeks.	1	15	Mechanic.	3 weeks.	4 weeks.	Occasional weather ache.	None.	None.	1 year.
31. T. P.[1]	1 day.	2.5	42	Athlete.	Over 5 months.	6 months.	None.	None.	None.	2 years.
32. A. R.	1 year.	2	19	Furnace stoker.	1 month.	2 months.	None.	None.	None.	3½ years.

[1] Case is discussed in text under "Analysis of Disability Time".
[2] Patient reports by letter.
[3] Case is discussed in text under "Postoperative Care".
[4] Patient had to care for invalid husband immediately after discharge.
[5] The wire suture was removed two years after operation.
[6] Patient was lost track of after six months.
[7] Unreliable record.

The Size and Shape of the Tear and the Amount of Retraction Involved

The size and shape of the tear, as well as the amount of retraction, varied tremendously. In view of the uniformity of the results, this is also interesting, since it has been stated by Bosworth[3] that disabling massive avulsions and old crescentic tears are doomed to fusion of the shoulder. The small tears have been found to recover function a little more rapidly, and the massive avulsions a little more slowly than the average lesions, but the overall results indicate that, assuming adequate repair along the lines of the technique suggested is carried out, the size of the lesion does not make any serious difference.

Postoperative Care

Proper convalescent regimen may be interfered with by various extraneous agencies, and the compensation status of any given case may be such that normal rehabilitation becomes delayed and complicated. Eight of the cases reported were under Workmen's Compensation, and their postoperative course was commonly complicated by such factors as unwillingness of the patient to cooperate fully, objections on the part of the employer or carrier to the patient doing any work until able to resume full duty, and—far from being least important—the insistence on the part of medical departments of the employer or carrier that the patient be treated by physical therapy as a postoperative regimen.

The patient in CASE 17 (Table II), F. P., was doing light jobs at two weeks, and at four weeks he was anxious and physically able to do his regular work. At that time he was examined by the medical department of the insurance company, and physical therapy was prescribed, with the result that he did not go back to his job until he had spent another month assimilating the doubtful benefits of an infra-red lamp thrice weekly.

In CASE 19 (Table II), J. F., the postoperative care, following the patient's discharge from the Hospital, was taken over by the carrier, and consisted of an intensive physiotherapeutic regimen. In spite of the fact that his tear was small and well repaired, eighteen months elapsed before he returned to his trade willingly, and most of this period was spent under treatment. At four months he returned for his first follow-up visit, stating that he was still completely disabled for his work as a carpenter, and with this estimation of disability, one was forced to agree, since the shoulder was stiff, atrophied, and painful from disuse.

The patient in CASE 24 (Table II), M. M., commenced his rehabilitation period under the handicap of having had an inadequate attempt at repair by a circular suture, which undoubtedly had something to do with the result. At any rate his postoperative care was taken over by an outside agency, and, in spite of intensive physical therapy over a long period, he did not recover for many months.

The patient in CASE 2 (Table I), P. G., had a massive avulsion. He remained under the supervision of the operator, and was back at his regular work in three months.

The patient in CASE 29 (Table II), T. B., remained under supervision. He was doing light work in one month, and returned to regular work in six weeks.

CASE 7 (Table I), G. H., was a massive avulsion and resulted in the dismal failure previously mentioned.

The patient in CASE 11 (Table II) remained under the supervision of the operator somewhat against the wishes of the carrier, whose medical department repeatedly tried to institute a regimen of physical therapy. He returned to light work in six weeks and resumed his regular job firing a furnace in two months.

CASE 14 (Table II), J. S. Although willing and able to return to light work at a much earlier date, this patient was not allowed to return to his job until complete rehabilitation had been accomplished.

There has been no set regimen for postoperative care. Physical therapy is useful when it is employed to help the patient progress in a program of active exercises, but is

[3] Patient reports by letter.
[5] Case is discussed in text under "Postoperative Care".
[6] Patient had to care for invalid husband immediately after discharge.
[7] Unreliable record.

considered to do much more harm than good when the patient gets the idea that the treatments are doing something active toward his rehabilitation. Under the latter circumstance, progress is almost always in reverse. Maintenance of mobility and gradual progressive resumption of use is the essence of the after-care, but only one person, the operating surgeon, whose judgment is tempered by a knowledge of the size of the lesion, the condition of the tissues involved, and the security of the repair, is qualified to evaluate the speed and completeness with which motion and use may be allowed. Immediate gentle motion in balanced suspension is the rule until the wound is healed. The patient is then allowed out of bed and is taught a program of active, gravity-free, pendulum exercises. As soon as symptoms and strength permit, wall-crawling and gibbet types of exercises are added. All exercises should be in short periods at frequent intervals, and should be done up to but not past the point of pain or fatigue. Five-minute exercise periods every hour on the hour are considered ideal. All patients are encouraged in the immediate resumption of normal light function, but as a rule active elevation of the extremity is not allowed until some time between the third and seventh week, depending upon conditions observed at the time of operation.

Fine stainless-steel wire was used for the main repair suture in two cases. Symptoms of bursal irritation developed in both more than a year after operation, and the resulting chronic bursitis did not subside in either case until after the metal sutures were removed. No. 3 Deknatel or No. 5 Pearsall silk was used in the remaining cases, and has been found to be very satisfactory for the purpose.

CONCLUSIONS

1. The transacromial approach is dry, easy, and especially useful for surgical procedures involving the cuff, capsule, humeral head, long biceps tendon, and intra-articular cavity of the shoulder.

2. Distinct benefits are gained by excising and discarding the outer fragment of divided acromion at the close of the operative procedure.

3. Retracted tears of the musculotendinous cuff can be repaired, according to the plan described, so that, regardless of their duration, size, shape, or amount of retraction, uniformly good results may be obtained.

4. Massive avulsion of the cuff, *per se*, does not warrant fusion of the shoulder.

5. The postoperative regimen following any cuff repair should remain under the control and close supervision of the surgeon who performed the operation.

REFERENCES

1. Bankart, A. S. B.: Recurrent or Habitual Dislocation of the Shoulder-Joint. British Med. J., II, 1132, 1923.
2. Bosworth, D. M.: An Analysis of Twenty-Eight Consecutive Cases of Incapacitating Shoulder Lesions Radically Explored and Repaired. J. Bone and Joint Surg., XXII, 369, Apr. 1940.
3. Bosworth, D. M.: The Supraspinatus Syndrome. Symptomatology, Pathology and Repair. J. Am. Med. Assn., CXVII, 422, 1941.
4. Codman, E. A.: The Shoulder. Rupture of the Supraspinatus Tendon and Other Lesions in or about the Subacromial Bursa. Privately printed. Boston, 1934. (Includes excellent bibliography prior to 1931.)
5. Davis, T. W., and Sullivan, J. E.: Rupture of the Supraspinatus Tendon. Ann. Surg., CVI, 1059, 1937.
6. Ferguson, L. K.: Suture of Ruptured Supraspinatus Tendon. Four Days after Injury. Am. J. Surg., XXIX, 294, 1935.
7. Fowler, E. B.: Rupture of Spinati Tendons and Capsule, Repaired by a New Operation. Illinois Med. J., LXI, 332, 1932.
8. Greeley, P. W., and Magnuson, P. B.: Dislocation of the Shoulder Accompanied by Fracture of the Greater Tuberosity and Complicated by Spinatus Tendon Injury. J. Am. Med. Assn., CII, 1835, 1934.

9. Haggart, G. E., and Allen, H. A.: Painful Shoulder: Diagnosis and Treatment with Particular Reference to Subacromial Bursitis. Surg. Clin. North America, XV, 1537, 1935.
10. Jones, Laurence: Reconstructive Operation for Non-Reducible Fractures of the Head of the Humerus. Ann. Surg., XCVII, 217, 1933.
11. Jones, Laurence: The Shoulder Joint—Observations on the Anatomy and Physiology. With an Analysis of a Reconstructive Operation following Extensive Injury. Surg. Gynec. Obstet., LXXV, 433, 1942.
12. Keyes, E. L.: Anatomical Observations on Senile Changes in the Shoulder. J. Bone and Joint Surg., XVII, 953, Oct. 1935.
13. Kocher, Theodor: Operative Surgery. p. 278–279. New York, William Wood and Co., 1894.
14. Mayer, Leo: Rupture of the Supraspinatus Tendon. J. Bone and Joint Surg., XIX, 640, July 1937.
15. Meyer, A. W.: 12. Bilateral Absence of the Tendon of the Long Head of the Biceps in One Subject, and Five Instances of Secondary Attachment of the Tendon to the Articular Capsule and Tuberosities, with Special Reference to the Effect of These Anomalous Conditions upon the Intertubular Sulcus. J. Anat. and Physiol., XLVIII, 133, 1914.

Anatomical Specimens of Unusual Clinical Interest. Am. J. Orthop. Surg., XIII, 86, July 1915.

Unrecognized Occupational Destruction of the Tendon of the Long Head of the Biceps Brachii. Arch. Surg., II, 130, 1921.

Further Observations upon Use-Destruction in Joints. J. Bone and Joint Surg., IV, 491, July 1922.

Evidences of Attrition in the Human Body (Abstract). Proc. Am. Assn. Anatomists. Anat. Rec., XXV, 142, 1923.

Further Evidences of Attrition in the Human Body (Abstract). Proc. Am. Assn. Anatomists. Anat. Rec., XXVII, 211, 1924.

Further Evidences of Attrition in the Human Body. Am. J. Anat., XXXIV, 241, 1924.

Spontaneous Dislocation of the Tendon of the Long Head of the Biceps Brachii. Report of Four Cases. Arch. Surg., XIII, 109, 1926.

Spontaneous Dislocation and Destruction of Tendon of Long Head of Biceps Brachii; 59 Instances. Arch. Surg., XVII, 493, 1928.

The Minuter Anatomy of Attrition Lesions. J. Bone and Joint Surg., XIII, 341, Apr. 1931.
16. Nicola, Toufick: Recurrent Dislocation of the Shoulder. Surg. Gynec. Obstet., LX, 545, 1935.
17. Nicola, Toufick: Recurrent Anterior Dislocation of the Shoulder. Its Treatment by Transplantation of the Long Head of the Biceps. Am. J. Surg., VI, 815, 1929.
18. Outland, T. A., and Shepherd, W. F.: Tears of the Supraspinatus Tendon. Résumé of Twelve Operated Cases. Ann. Surg., CVII, 116, 1938.
19. Smith-Petersen, M. N.; Aufranc, O. E.; and Larson, C. B.: Useful Surgical Procedures for Rheumatoid Arthritis Involving Joints of the Upper Extremity. Arch. Surg., XLVI, 764, 1943.
20. Toumey, J. W., and Haggart, G. E.: Surgery of the Shoulder Joint. Surg. Clin. North America, XXII, 883, 1942.
21. Wilson, P. D.: Complete Rupture of the Supraspinatus Tendon. J. Am. Med. Assn., XCVI, 433, 1931.

DISCUSSION

Dr. M. N. Smith-Petersen, Boston, Massachusetts: Last fall several of us had the good fortune to see many of the cases that Dr. McLaughlin has discussed; they were uniformly good. I like to emphasize this point since Dr. McLaughlin has not emphasized it enough.

Acromioplasty in connection with the operation which Dr. McLaughlin has described, is well worth while. We have used it in a fair number of cases of rheumatoid arthritis with very satisfactory results. It eliminates pain arising from the subacromial bursa.

There are two points that I would like to bring out about the procedure of acromioplasty:

1. The acromion must be excised up to, but not including, the acromioclavicular joint. There should be sufficient bone on the acromial side of this joint to prevent any interference with its function.

2. In regard to repair, the periosteum is incised between the attachment of the trapezius and the origin of the deltoid. In repairing the defect created by the excision of the acromion, the central tendon of the trapezius is sutured to the periosteal attachment of the trapezius. By this means the sulcus at the point of the shoulder is diminished but not eliminated.

Dr. D. M. Bosworth, New York, N. Y.: What I have to say is not critical of Dr. McLaughlin's work. I think perhaps he may have misinterpreted the things he has seen, or what I have said. In the first series of diagrams, laceration of the supraspinatus alone was shown. I do not think any of us will have a great amount of

difficulty in securing excellent results in those cases, if we remember that the corner of the supraspinatus is turned back and retracted. If you have full-thickness tears of only part of the cuff, you repair them easily, and the results will be good. In repair of old lacerations of the tendon, the result is not so good, because you have poor tendon material plus adhesions. If you take out a part of the articular cartilage and suture the tendon to the head of the humerus, you are bound to lose some motion.

Dr. McLaughlin's incision is the same as cutting the deltoid close to the acromion and then chipping off the acromion. If you cut the deltoid close to the acromion, you can do a lot of work without taking off the acromial margin.

In complete avulsions of the cuff, I believe primary fusion of the shoulder is the best repair. Dr. McLaughlin's cases did not show complete avulsion. Where you have complete avulsion of the cuff, it will be turned in between the head of the humerus and the glenoid. His did not show any turning in or any case of rupture of all four tendons. Those tears are not found in young persons, but always in old ones with osteoporosis. In one of my cases, one repair held until the operative site was out of view; then it pulled out. In another one, the silk sutures ripped through as though the bone were cheese. If repair of complete avulsion in old people with osteoporotic bone is attempted, it will not stand up.

Poliomyelitis patients with rotator paralysis can initiate abduction, for the acromion does not impinge against the upper border of the greater tuberosity of the humerus. I have seen that done repeatedly, and with fairly good force. Reattachment of one or more tendons will enable the patient to abduct. Active abduction also can take place in cases where there was never a repair. I have opened shoulders and found total laceration of the cuff, and the men had been working for many years. Fowler has shown many shoulders with avulsion of the cuff in patients who carried on without too much difficulty. In these patients, the upper border of the tuberosity wears off and no longer impinges.

In the cases I have repaired, there have been some that completely healed and then later the patient complained of pain. I classified those as due to the wearing out of the already damaged tendons. As time goes on, therefore, one finds more cases unsatisfactory.

DR. PHILIP D. WILSON, NEW YORK, N. Y.: I had the pleasure of seeing Dr. McLaughlin present a number of his cases last December, and I can testify to the excellent functional results in the patients that he showed. I have been interested in tears of the tendon cuff for a number of years, and formerly was very enthusiastic about repairing some of them, but I lost my enthusiasm as I had more of the chronic cases,—those tears which had been present for four months or longer. I had not solved the secret of the triangular tear, as Dr. McLaughlin has, and I was trying to pull the apex of the tendon straight out to the tuberosity. I found out that I could not close the gap. Seeing Dr. McLaughlin's demonstration has renewed my interest in the subject, and I have operated upon several patients since that time.

I think the transacromial approach has a great degree of usefulness in operations upon the shoulder. Dr. McLaughlin himself observed that it was possible to use this incision for doing a Nicola repair for recurring dislocation of the shoulder. I had a case last year in which I had to use the incision because of an acne over the shoulder joint, through which I was afraid to make an incision. I found it quite possible to retract the lateral flap and to do the regular Nicola procedure. It leaves a much better scar than the deltoid-splitting incision, and, if it is sutured with stainless steel, there is no trouble in obtaining a good repair of the bone.

I have not done excision of the acromion. I think the point made by Dr. Smith-Petersen is very important, and is one that we ought to follow. I am going to try it.

In conclusion, I would like to point out that the diagnosis of tendon rupture at the shoulder is often very obscure. Last winter I had a man of seventy come in. He simply had a sore and painful shoulder. His pain had begun one morning on getting up. There was absolutely no history of trauma. He was an emaciated, very thin man, with a soft fluctuant swelling under the deltoid. I aspirated and obtained sixty centimeters of old blood. This argued for either a neoplasm, or tuberculosis, but most likely a neoplasm. The roentgenogram was negative, except for a large soft-tissue shadow. I finally came to the conclusion that the patient had a tear of the tendinous cuff. I explored and found a complete avulsion of the entire cuff. It was not feasible to repair it in the usual way, so I took off the head of the humerus and sutured the tendons to the stump of the neck. Of course he has not obtained a large amount of function, but it is stable and free from pain. He can abduct his arm 45 degrees, and he is very happy about the result.

DR. J. ALBERT KEY, ST. LOUIS, MISSOURI: May I ask one question. In two ruptures which I could not repair, I pulled down the tendon as far as I could and cut it off, letting it retract. The results were fair. I would like to ask Dr. McLaughlin if he has had experience with this method.

DR. SAMUEL KLEINBERG, NEW YORK, N. Y.: I appreciate that it has been the generally accepted opinion that the supraspinatus initiates abduction at the shoulder. From my clinical experience I have reason to doubt it. I have always been impressed by two observations: First, in almost all of my cases of acutely painful shoulders, the patient always had a small range, usually 20 to 30 per cent., of voluntary abduction, even when rotation inward and outward were completely restricted. Second, in cases of severe and persistent shoulder disabilities that did not yield to conservative measures, and in which I felt obliged to operate, particularly in those instances in which the roentgenogram showed extensive calcareous infiltration of the supraspinatus, excision of the supraspinatus tendon did not militate against full functional recovery.

About ten days ago, I operated upon a man who had a very marked disability of the shoulder of many months' duration. He had a very extensive calcareous deposit in the supraspinatus tendon. I excised the calcareous material and the supraspinatus tendon, making no attempt at repair of the excised tendon. Within three days the patient began using his shoulder and before I left New York, the other day, he had almost complete abduction of the operated member. I am, therefore, convinced that the rôle of the supraspinatus as an abductor of the shoulder is a very minor one, and that only in exceptional, acute traumatic lesions does a tear of this tendon require surgical repair.

Dr. Harrison L. McLaughlin, New York, N. Y. (closing): The problem of deformity following acromioplasty, mentioned by Dr. Wilson and Dr. Smith-Petersen, has been most easily overcome by beveling off and reshaping the remaining stump of the acromion and snugly reattaching the separated deltoid origin to the tissue overlying the stump.

Dr. Bosworth remarked that no difficulty has been encountered in working under an intact acromion, but our experience has been that many lesions are extremely difficult, if not impossible, to repair adequately, without obtaining the added exposure gained by a division of this bone.

The avulsion illustrated represented the mean between the extreme and the mild degrees of pathology. As I remember it, Dr. Bosworth's article prescribed arthrodesis for all complete avulsions and old crescentic tears. As far as scar tissue is concerned, the present series includes old tears with peripheral cicatrization, and more recent tears that had occurred through tissue weakened by pre-existing infiltrative changes. The peripheral scar tissue in both types requires excision before healthy viable edges can be reapproximated and uneventful healing can be anticipated. Few actual adhesions have been found.

There is full agreement with the remarks of Dr. Kleinberg, Dr. Bosworth, and Dr. Key to the effect that all cases do not require repair. Of the three thousand odd patients in this series who complained of sore shoulders, many must have had tears, but only seventy-five were characterized by disability or symptoms sufficient to warrant repair.

Dr. Wilson's remarks on the use of the transacromial approach deserve emphasis. It has been used for the Nicola operation. Reattachment of the anterior capsule to the glenoid for recurrent dislocation has been done through this approach with ease and without the added trauma caused by transverse section of the coracoid and subscapularis. Through this approach, fragments of the dislocated humeral head have been removed from above, rather than through the brachial plexus, and the inevitable soft-part tears accompanying complicated fracture-dislocations have been much easier to repair with the whole pathological field in full view.

BLADE-PLATE INTERNAL FIXATION FOR INTERTROCHANTERIC FRACTURES

BY AUSTIN T. MOORE, M.D., COLUMBIA, SOUTH CAROLINA

In recent years, comparatively little has been written concerning the treatment of intertrochanteric fractures. The purpose of this article is to present an appliance which has proved itself to have decided advantages over older methods of treatment. By this method practically all intertrochanteric fractures and fractures of the upper femoral shaft can be satisfactorily treated.

It seems to be a common understanding that, due to the rich blood supply of this area, intertrochanteric fractures can be treated by almost any method. This is not true. The author has seen cases of non-union following fractures through the base of the femoral neck, through the trochanters, through the subtrochanteric region, and through the upper femoral shaft. A varus deformity with shortening of the extremity is frequently seen after intertrochanteric fractures. In addition to the above complications, the usual traction treatment of these fractures requires prolonged hospitalization, expensive nursing care, and several months for convalescence. The patients, as a rule, are not comfortable; pressure sores may develop; and joint stiffness is seen frequently. In elderly patients, the outcome is not infrequently fatal.

Russell's traction, skeletal traction and counterpoised suspension, well-leg traction, and other methods have been improvements over the older methods of Buck's traction or a plaster splint. None of these methods approaches the fracture directly, and none provides immediate reduction and rigid fixation of the fracture, with early ambulation and resumption of normal function. Various methods of internal fixation have been tried, and protruding pins have been used to hold the fragments in position.

Roger Anderson not only developed his well-leg traction device, but has described his method of pin fixation for these fractures. Lawson Thornton in 1937 described

FIG. 1-A

FIG. 1-B

Showing method used in 1934. C. D. S. had a comminuted intertrochanteric fracture which was treated with a single Moore nail and bolt. No external fixation was necessary. (The roentgenograms have been retouched.)

FIG. 2

Showing method used in 1935. Mrs. J. G., eighty years of age, had an intertrochanteric fracture which was treated with four Moore nails. (The roentgenogram has been retouched.)

FIG. 3-A

Showing method used in 1938. The aluminum framework was made to support rigid threaded pins used in treating intertrochanteric fractures. The lower pin is a special "removable bolt" which prevents shifting of the bone from side to side. This method was cumbersome, difficult to apply, and was soon abandoned.

FIG. 3-B

Mrs. J. G., aged seventy-five years, had an intertrochanteric fracture treated with pins in an aluminum framework. Solid union in perfect position was obtained ten weeks after the accident.

FIG. 4

Single-angle blade-plate for intertrochanteric fractures.

his extension plate to the Smith-Petersen nail, and later Alonzo Neufeld introduced his appliance made of channel steel.

In 1934, the author began to treat intertrochanteric fractures by open reduction and internal fixation. The earlier cases were treated with several Moore adjustable hip nails placed at the best mechanical advantage. At times one or more small bolts were used in addition to the nails. The bolts were fashioned from the nails by using an additional nut on the nail. The protruding end of the bolt was cut off with wire nippers. Most of the intertrochanteric fractures could be treated by this method, but at times the internal fixation was inadequate, and, in addition to the internal fixation, some form of external support was necessary.

In 1938, a method of pin fixation after open reduction was devised by the author. The pins were large and threaded; they protruded from the flesh and were held in place in a metal framework. This method assured perfect reduction, rigid immobilization, and early resumption of functional activity. The results were satisfactory, but the metal framework was awkward and hard to apply, and the uses of metals of different electrical potentialities caused pin-wound seepage and irritation. The method was soon abandoned.

After several years' experience, the use of multiple nails, bolts, or protruding pins was found to be inadequate fixation for many cases of intertrochanteric fractures. In 1940, the author began to use an appliance which held the upper fragment by a blade in the head and the lower fragment by a plate on the shaft of the femur. Fractures of the upper femoral shaft could also be treated by this method. The appliance was similar in principle to the nails of Thornton and Neufeld. The appliance was made by our brace maker from the concave stainless-steel brace material which he used in making his braces. The appliance was approximately eight inches long and angled to 135 degrees to correspond to the angle of the neck on the shaft of the femur. The upper three inches, or blade portion of the appliance, was sharpened at its upper end so that it could be driven up through the neck and into the head of the bone. The lower five inches extended along the outer side of the shaft of the femur and was held in place by screws. The concave shape of the appliance assured a good grip of the blade portion in the head and neck of the bone, and prevented any side-slipping. It also allowed the plate portion to fit snugly

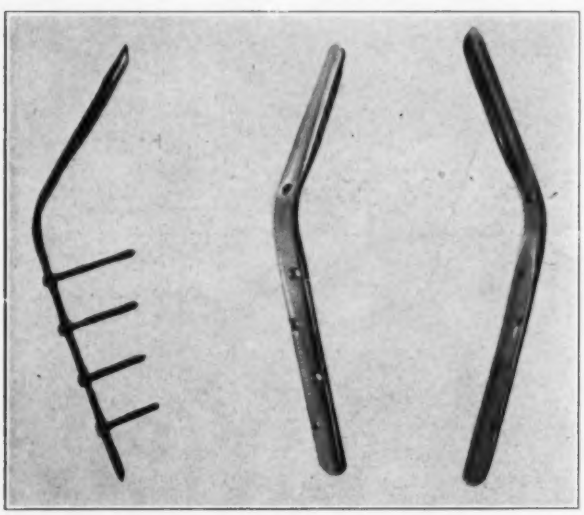


FIG. 5

Three views of the single-angle blade-plate. The blade is concave to secure a better grip on the head, and the plate is slightly concave to fit snugly against the femoral shaft. At the bend, the appliance is straight rather than concave, so that its shape can be changed if necessary. For added strength the bed of the angle is thickened. The point of the driver-extractor is engaged in the drill hole just above the angle. Drill holes along the shaft are staggered to secure better fixation with screws. In the illustrated model, the top drill hole on the shaft should be higher and the lower drill hole should be at the bottom of the appliance.

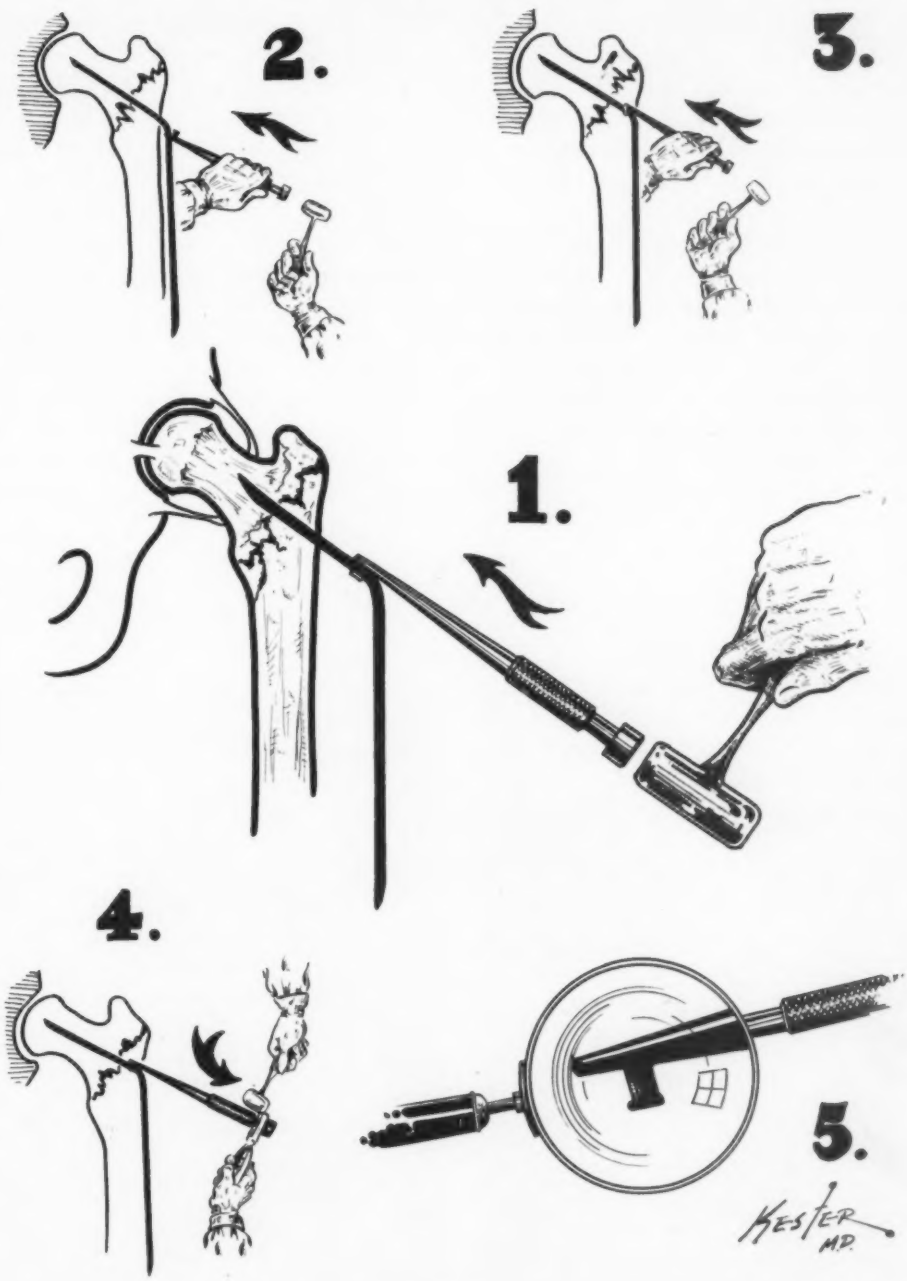


FIG. 6

1: The blade-plate is driven in with hook of driver inserted in the hole above the bend. A small projection on the driver hook locks the driver to the blade-plate.

2: To seat the blade, the driver point may be inserted in the hole below the bend.

3: The blade may be seated with the driver in hole above the bend. In this position, the outer thin bony cortex is penetrated,—a matter of small consequence.

4: The blade-plate is extracted by the hook of the driver being placed in the hole above the bend. A pair of pliers grips the outer end of the driver. Extraction is not difficult with light blows of a mallet.

5: The point of the driver-extractor has been magnified to show the anterior projection on the lower portion of the hook, which locks the apparatus in place when it is used as a driver. The posterior portion of the hook is under-cut or angled to prevent the hook from slipping out of the hole in the blade when the appliance is used as an *extractor*.

against the side of the shaft of the femur. A driver-extractor for the appliance was made from a small rod of stainless steel, bent at its end in the shape of a blunt hook. This hook fits into a hole in the appliance just above the bend of the angle, and through it the appliance can be either driven in or extracted. If desired, the blade of the appliance can be driven in partially with the driver in the hole above the bend and finally seated by shifting the driver to a hole just below the bend of the appliance. The driver is fashioned with a pointed end for this purpose.

Due to the concave shape, and for the lack of a better term, the appliance was first called "goose-neck appliance". Blount of Milwaukee suggested modifying the appliance by bending it at another angle—thus making a true "goose-neck"—and using this modification for internal fixation of subtrochanteric osteotomies. This proved to be very efficacious. He also suggested the name "blade-plate". The author and he agreed to describe the appliances as blade-plates,—the single-angle blade-plate for intertrochanteric fractures, and the double-angle blade-plate for subtrochanteric osteotomies. Blade-plates with flat and with gouge-shaped blades have been used. A preference is expressed for the gouge-shaped blade. The appliance can be shaped in a variety of ways for various purposes.

Two of the blade-plates of brace steel have been removed after remaining in place approximately two years. The brace material proved to be of an inferior quality of stainless steel, and when these two appliances were removed, there was evidence of mild

FIG. 7

J. K. B., aged sixty-seven, had a severely comminuted intertrochanteric and upper femoral-shaft fracture which was treated with a single-angle blade-plate with a six-inch plate. (The roentgenogram has been retouched.)

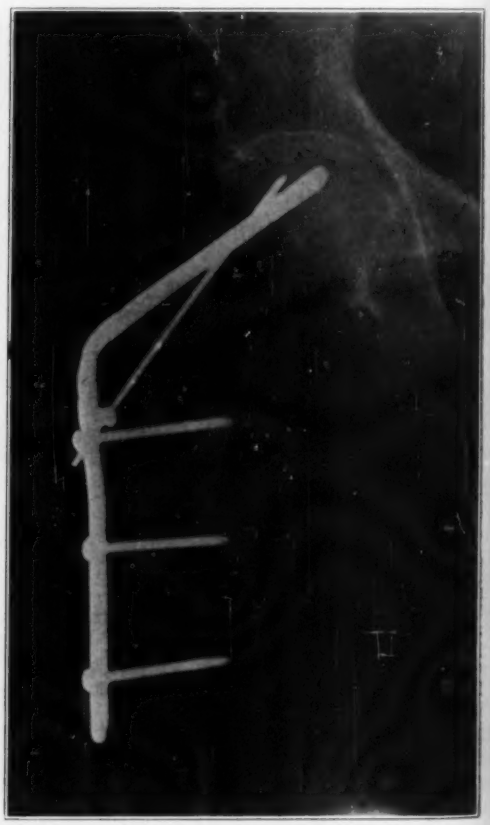


FIG. 8

A. M. H., aged fifty-eight, had an intertrochanteric fracture which was treated with a single-angle blade-plate. A large loose fragment of the intertrochanteric ridge was held in place with an additional Moore nail.

FIG. 9-B

FIG. 9-A

Fig. 9-A: Showing a healed intertrochanteric fracture, which had been treated with a Thornton hip nail, and a severely comminuted refracture of the upper femoral shaft.

Fig. 9-B: Result following removal of the Thornton nail, reduction of the fragments, and fixation with a long single-angle blade-plate. Additional fixation was provided by inserting in the femoral shaft four pins, the ends of which were embedded in an external plaster bar. This patient died of pneumonia ten days after operation.

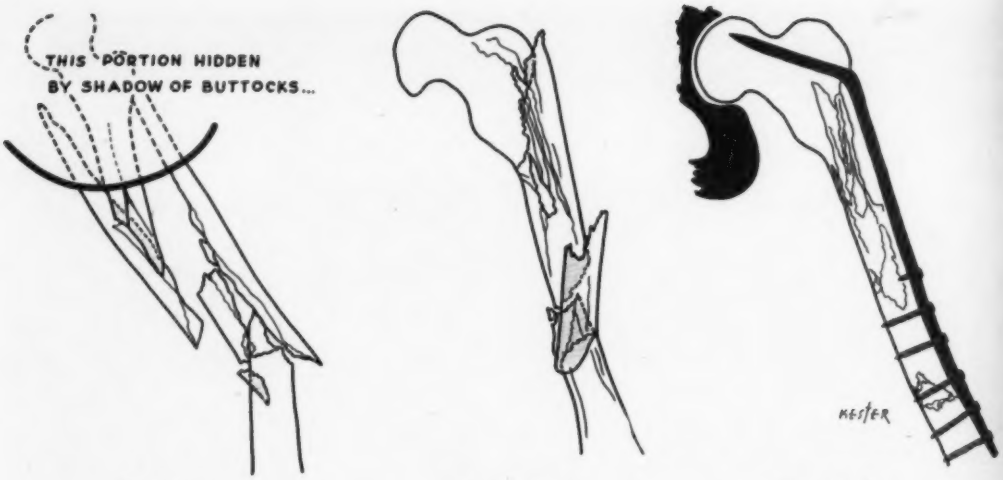


FIG. 10-A FIG. 10-B FIG. 10-C

W. L. The first two views show the anteroposterior and lateral views of a severely comminuted fracture of the femoral shaft. Replacement was impossible with skeletal traction. The last view shows a specially made blade-plate, with the plate approximately twelve inches long, extending from hip to knee joint.

corrosion. The blade-plates are now available commercially, and they are being made of standard quality S.M.O. 18–8 stainless steel. Two more-or-less standard sizes have been used,—two and one-half inch blades with four and one-half inch plates, and three-inch

FIG. 11

M. R., aged seventy-three years, had fractures of both hips. An intracapsular fracture on the left in 1940 was treated by four Moore nails. An intertrochanteric fracture on the right in 1942 was treated with a single-angle blade-plate. The patient regained full functional recovery in both hips.

FIG. 12-A

FIG. 12-B

N. G., aged ninety-three years, had a high femoral-shaft and subtrochanteric fracture treated with special blade-plate with extra long plate. (The roentgenograms have been retouched.)

blades with five-inch plates. For fractures of the high femoral shaft, a six, seven, eight, or nine-inch plate may be necessary, and a three and one-half inch blade at times may be desirable. Any of these sizes may be obtained. In one patient, who had a severely comminuted fracture of the femur which could not be realigned by skeletal traction, a blade-plate was used that extended from the hip to the knee joint.

The blade-plate has many advantages. It is simple in construction, and it can be applied without difficulty. It is strong enough to maintain position of the fragments with moderate weight-bearing. The operation can be done without shock; no external support is necessary; and, as a rule, the patient may leave the hospital, walking on crutches within two weeks after the operation. Without obtaining permission, one of our patients went back to work with no external support three weeks after operation. Graduated weight-bearing is allowed, and full weight-bearing is restricted until roentgenograms demonstrate solid bony union. At the time of operation, the appliance may be bent, or its shape may be changed, with small bending irons which have been sterilized for this purpose.

Since 1934, eighty-three cases of intertrochanteric fractures have been treated by open reduction and internal fixation. The blade-plate has been used in forty-one of these cases. There have been no operative deaths and no cases of non-union. There was one minor wound infection.

TECHNIQUE OF OPERATION

As a rule, the fragments can readily be reduced, but at times reduction is very difficult, and complete muscle relaxation is necessary. For that reason, a low spinal or pentothal anaesthesia is preferred for the operation. The patient is prepared from the waist to the

FIG. 13-A

FIG. 13-B

C. O., aged thirty-four years, had a comminuted subtrochanteric fracture. (The roentgenograms have been retouched.)

toes on the affected side. The extremity is wrapped in sterile dressings, lies free on the table, and is easily manipulated. Underneath the patient is a tunnel for roentgenograms. The head of a mobile roentgenographic unit is positioned directly over the affected hip,

FIG. 14-A

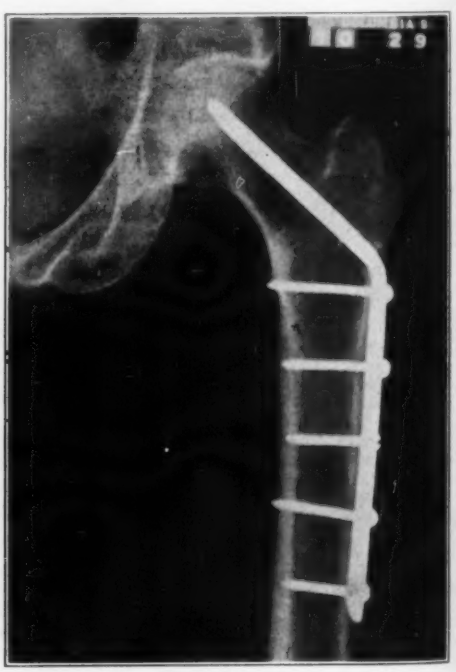


FIG. 14-B

S. D. W., aged sixty-five years, had a comminuted intertrochanteric fracture. She walked with the aid of crutches five days after the operation.

FIG. 15

E. W., aged sixty-seven years, had a comminuted intertrochanteric fracture. (The roentgenogram has been retouched.)

FIG. 16

F. P. B., aged sixty-three years, had a nonunion of the fractured neck of the femur of five months' duration. She was treated with a Blount-type double-angle blade-plate, made of concave brace steel. Bony union resulted in six months with full functional recovery.

and the x-ray machine is not moved again during the operation. The first roentgenographic exposure is made immediately after manipulation of the limb for reduction. The roentgenograms are developed in a dark room adjoining the operating room. Warm concentrated solutions are used, and the roentgenograms should be ready for inspection within three minutes. The operative incision is made, and the wound is draped while the first roentgenogram is being developed. The incision extends from the trochanter, down the outer side of the thigh, usually for five to six inches. Lateral roentgenograms may be made by the use of the lateral or curved casette. A guide pin may be inserted at the vasternus line and driven up through the neck and into the head. Check-up anteroposterior and lateral roentgenograms may be made with the pin in place. It is the author's custom to make the lateral (inferior superior) view by flexing the thigh to less than a right angle, and holding it there in abduction and external rotation. This position gives a full-length unobstructed view of the neck and head of the bone. With experience, the surgeon can select a blade-plate of the proper size and insert it without a guide pin. When this is done, the second set of roentgenograms is not necessary. A Lowman bone-clamp holds the plate to the shaft of the femur while the anteroposterior and lateral views are taken. While the roentgenograms are being taken and developed, the plate is usually anchored in place with screws. The electric motor and a special, smooth-shank, stainless-steel drill that will not break are used to make the drill holes. With the use of a depth finder, screws of a proper length are selected.

When the blade is inserted without a guide pin, it enters the bone through a small osteotome cut on the outer surface of the shaft approximately three-fourths of an inch below the vasternus line. This line is the line of origin of the vastus externus, and is the

base of the greater trochanter. With the limb in internal rotation, the blade is driven in parallel to the horizontal table top, and it is directed toward a point midway between the anterior superior spine of the ilium and the spine of the pubis. With these landmarks in mind, placement of the blade is not difficult. The length of the blade is selected by comparing it with the roentgenogram taken at the beginning of the operation. As the x-ray is a projected image, the length of the neck as it appears on the film is longer than normal. The blade length required is usually about three-fourths of an inch shorter than the x-ray image. By the sense of feel, the blade can be driven up through the neck and into the head. If hard cortical bone of the neck is encountered, the blade is redirected. The driver is also the extractor. By tapping on a pair of pliers that grasps the driver just behind its enlarged head, the blade is easily extracted when necessary. The sharp cutting edge of the blade allows it to be driven in with light blows of the mallet. The blows of the mallet are not sufficient to cause damage to the articular cartilage, and the dangers of aseptic necrosis and traumatic arthritis are minimized. The upper dense weight-bearing portion of the head is felt when it is reached. By rotating the extremity, one easily determines that the joint has not been penetrated. The straight sides of the blade are a decided advantage. If the neck should be missed, or if the blade emerges from the neck, immediately the sense of resistance is lost and the blade is redirected. The head of the bone is usually located by inserting a pin by hand before the blade is driven in place.

Although speed is not a proper criterion by which to judge any operation, a speedy operation is frequently of great importance to many aged and debilitated individuals with intertrochanteric fractures. The type of operation described can be done comfortably in twenty to twenty-five minutes. In cases where there is comminution of the fragments, a proportionately longer time for operation is required.

SUMMARY

1. A blade-plate is presented for the treatment of intertrochanteric fractures and those of the upper femoral shaft.

2. The length and shape of the appliance may be altered, and the principle of treatment may be applied to a variety of conditions.

3. By this means of treatment, convalescence is shortened, expense of hospital and nursing care is reduced, and a full functional recovery may be expected.

SULFONAMIDES IN THE TREATMENT OF CHRONIC OSTEOMYELITIS*

BY J. ALBERT KEY, M.D., ST. LOUIS, MISSOURI

From the Department of Surgery of the Washington University School of Medicine, St. Louis

In the earlier days of sulfonamide therapy, the results of the treatment of bone infections were disappointing because sulfanilamide and sulfapyridine were the drugs in general use, and these have relatively little effect upon staphylococcic infections. The introduction of sulfathiazole and sulfadiazine has altered the picture, because the growth of both streptococci and staphylococci is inhibited by concentrations of these drugs, which may be maintained in the blood stream with relative safety.

Bacteria which are free in the blood stream are especially vulnerable to the drug, when it is administered systemically. Organisms in the tissues, in areas of inflammation, and in abscess cavities are less vulnerable to the sulfonamides for several reasons:

1. Actual contact of the drug with the bacteria is necessary, and the effectiveness of the drug varies directly with its concentration. The concentration in the tissues is less than that present in the blood stream (about one-half), and it is probable that many bacteria in inflamed areas are so well walled off from the blood stream that very little of the drug reaches them.

2. The action of the sulfonamides is inhibited by products of tissue destruction (peptones). The drugs are less effective if large numbers of bacteria are present.

3. In low concentrations, the drugs do not kill bacteria, but merely inhibit their growth, and they are then destroyed by the defense mechanisms of the host. However, inhibited but still living organisms may remain in the tissues, escape destruction, and later become active.

It is thus evident that the sulfonamides in the blood stream cannot exert their maximum effect upon bacteria in localized areas of disease; this is especially true in chronic osteomyelitis where some of the bacteria may be thoroughly isolated from the blood stream, and located in areas where the leukocytes cannot reach and destroy them, even if their growth is inhibited. On the other hand, we now have sufficient clinical evidence to warrant the conclusion that the systemic administration of sulfadiazine, and especially of sulfathiazole, to patients with acute osteomyelitis or an acute exacerbation of chronic osteomyelitis, usually has a beneficial effect, not only upon the septicaemia, but also upon the local disease.

There is, however, considerable difference of opinion as to how much reliance is to be placed upon the drug in the treatment of the local disease. Some surgeons (Hoyt, Davis, and Van Buren) believe that most cases of acute osteomyelitis can be cured by the systemic administration of sulfathiazole, and that operation is rarely necessary. Others, of whom the author is one, believe that sulfathiazole is a valuable adjunct in the treatment of this disease, but that the focus in the bone should be drained and the limb immobilized as soon after the onset of the disease as the patient can stand the relatively minor operative procedure. Failure to do this may result in increased destruction of bone.

In chronic osteomyelitis, continuous administration of the drug over a long period will lessen the amount of discharge, but it will not cure the disease, because it cannot sterilize dead bone or cavities with necrotic contents and rigid walls. Here the problem is largely a mechanical one of removing the dead and infected bone, of eliminating the dead spaces, and of covering the remaining living bone with soft tissue that has an adequate blood supply to permit healing. In the Orr treatment, the operation was performed in an effort to obtain these results, the wound was packed loosely with vaseline gauze, left wide

* Read at the Annual Meeting of The American Orthopaedic Association, Cleveland, Ohio, June 7, 1943.

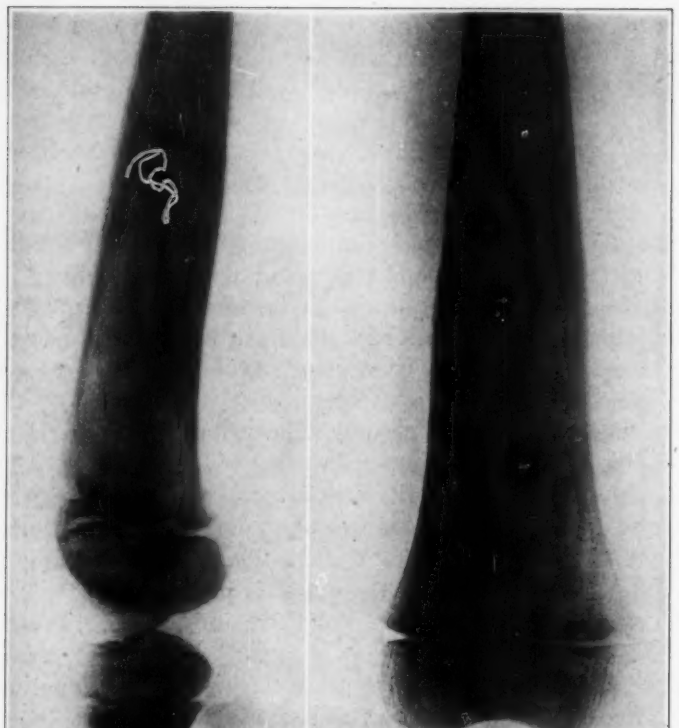


FIG. 1

Medium-sized cavity, with sequestrum and sinus, was excised after eighteen months. Healing by primary intention followed closure.

open, and permitted to heal by granulation. For several years the author has been partially closing such wounds, leaving in small vaseline wicks for drainage. This has resulted in quicker healing and in better scars. He has also used various chemicals in the wounds, and, at the American College of Surgeons in St. Louis in 1932, he showed cases in which the wounds had been almost completely closed and drained by wicks of gauze impregnated with merthiolate ointment. A review of these experiments indicated that neither the merthiolate ointment nor any other of the antiseptics used was any more effective than plain vaseline.

In 1941, Dickson, Diveley, and Kiene reported a series of twenty-two cases of chronic osteomyelitis in which the infected soft tissue and bone had been meticulously excised, the wound dusted with sulfathiazole powder and closed in layers, and the extremity immobilized in a plaster cast. The patients were given sulfathiazole by mouth for five days before the operation and for about fifteen days after the operation. Healing by primary intention occurred in 82 per cent. of the patients.

The author began using their method in 1940, after Dr. Dickson told him of their first success, and he has used it in operating upon 101 consecutive patients with chronic pyogenic infection of bone. Four patients were operated upon twice, two were operated upon three times, and one was operated upon four times. Of the 101 patients, the infection was in the tibia in thirty-three, in the femur in twenty-four, and in other bones in forty-four. In four patients, at the first operation, an acute abscess of an old chronic osteomyelitis was incised and drained, and no attempt was made to close the wound. In one of these, amputation became necessary later, and in the others healing has now taken place. The remaining ninety-seven patients were all operated upon while the disease was relatively quiescent,—that is, the patient was afebrile, and there were no general toxic symptoms and no evidence of acute infection in the local lesion. In these patients, an attempt was made to close the wound at the primary operation. This was not always possible, because of lack of soft tissue to cover the bone.

The technique followed was similar to that used by Dickson, Diveley, and Kiene, except that the excision of the infected scar tissue and sinuses was not carried out so meticulously, and, in several instances, extensive subfascial and deep abscesses were curetted and collapsed rather than excised. The usual saucerization operation was performed on the abscess cavities in the bone, and an effort was made to remove all sequestra and to eliminate dead spaces. However, no effort was made to remove all eburnated bone,

which we believe is chronically infected and may contain minute abscess cavities which are not visible in the roentgenogram. As few vessels as possible were ligated, and these with fine silk, and no deep sutures were used. The skin and subcutaneous tissue were closed in one layer, usually with a continuous suture of silkworm gut, after the wound had been sprinkled liberally with sulfathiazole powder. Then a pressure dressing was applied, and usually the limb was immobilized in a plaster cast.

Stainless-steel wire or screws were used in four instances, and in each instance the wound healed; but later a small sinus developed and the offending wire or screws were removed.

In reviewing the results obtained in these patients, it is evident that each chronic pyogenic lesion in bone presents an individual problem which must be solved by surgical principles, and cannot be solved by chemotherapy. On the other hand, the use of chemotherapy has considerably increased the safety of our surgical procedures and has enabled us to close a large percentage of the wounds after a radical operation for the cure of chronic bone infection.

In this connection, it is well to consider the dangers of chemotherapy. In this series, the adult patients were given sulfathiazole in doses of from one gram every six hours to one gram every four hours on the day before the operation or earlier, and this was continued for about a week after the operation. Children were given the drug in smaller doses according to their body weight. In addition, from two to five or even more grams of the drug was placed in the wound before it was sutured. Routine blood levels of the drug were not determined, but the fluid intake and output were watched, and fluids were forced to a moderate degree. One patient with severe renal damage was not given the drug by mouth because he could not tolerate it. However, about five grams of sulfathiazole was sprinkled in the wound when his hip was disarticulated, and he made an uneventful recovery. The generalized oedema subsided promptly, and his condition improved after the infected femur had been removed.

FIG. 2

Excision of third metatarsal shaft and part of tarsal bones, after three months, was followed by primary healing.

Other than postoperative fever, which may or may not have been due to the drug and which in no instance was alarming, toxic reactions occurred in only six patients. These included suppression of urine in one, hematuria in one, and skin rashes in four. All subsided promptly when the drug by mouth was stopped and fluids were forced. It is believed that these were due to the drug administered by mouth, because in over 600 consecutive clean operations in which sulfanilamide or sulfathiazole powder or a mixture of the two drugs was implanted in the wounds there were no toxic reactions other than fever, and only two mild infections following operation (one from a lymph accumulation and one from a hematoma).

In only one instance in this series was it necessary to open the wound on account of an abscess after the operation. This was in an arthrodesis of an old tuberculous hip which was secondarily infected. Bony union was obtained, but the sinuses are still

FIG. 4-A

Showing large and medium-sized cavities with sinuses of seven years' duration.

FIG. 4-B

Healing was by primary intention after excision and closure.

FIG. 3

Sequestrum and medium-sized cavity, present for ten years, were treated by excision and closure. Healing was by primary intention.

FIG. 5-A

Old compound fracture with severe infection of one year's duration.

FIG. 5-B

Astragalectomy with closure failed, and amputation was necessary.

draining. In two other cases in this series, tuberculosis was present and was complicated by severe pyogenic infection. One knee with extensive abscesses in the popliteal space and posterior thigh was fused, and the sinuses were healed when the cast was removed twelve weeks after the operation. In the other case, the disease involved the first metacarpal and adjacent areas of the hand and wrist. Extensive excision, with amputation of the thumb and partial closure, resulted in a granulating wound which is healing slowly. It has been the author's experience that the sulfonamides have no influence upon tuberculous infection, but they are useful in a mixed infection.

There was one death in the series. This patient was a woman, eighty-seven years old, who had fractured a hip about a year before. The hip had been nailed, but the head of the femur was dead, the neck had been absorbed, and the distal fragment had slipped up on the nail. She returned to the Hospital with an ulcer and abscess over the projecting head of the nail, the infection extending into the hip joint. The nail and dead head were removed, but the patient gradually became weaker and died about two weeks later.

The series includes eleven amputations of one lower extremity; all were in adults, with infection so extensive that excision was considered impractical, as the patient would be left with a foot and ankle which were worthless from a functional standpoint. Five had non-union, and two had old compound fractures at the ankle with extensive chronic infection. The author performed astragalectomies on these two and they did very well after the operation. However, when the casts were removed, it was obvious that amputation was necessary, and this was done without wasting any more time. In all of the amputations, the wounds were closed without drainage, and healed by primary intention.

Including the eleven amputations, sixty of the wounds healed by primary intention, but sinuses later developed in four of those which contained metal, and the metal had to be removed. Amputation was necessary in one of these cases; while in the other three healing occurred. In almost all of the other cases healing has taken place. The tuberculous hip is still draining after one year, and a femur with a large dead space is still draining after six months. Another femur in which amputation was advised is still draining, and infected bone surrounded by extensive scar tissue is exposed on two tibiae.

All of the failures have been in adults. This is largely because children's bones have more regenerative capacity than do adult bones, and it is possible to perform a more radical operation for a given area of infection and still obtain a useful extremity. Also, in children, if the main focus is fairly well excised, the adjacent bone which is mildly infected will frequently heal, and dead bone which is not sequestrated will be absorbed and replaced by living bone through creeping replacement. This is especially true in young children and in cancellous bone.

FIG. 6-A FIG. 6-B

Fig. 6-A: Compound, comminuted fracture with infection and comminution of seven months' duration.

Fig. 6-B: Excision and closure, with bone graft and multiple drilling, were followed by primary healing. Wound is now healed and fracture is united.

It is evident that the percentage of primary healing in this series is less than that obtained by Dickson, Diveley, and Kiene. However, their series apparently represents a selected group of cases in which it was possible to perform a satisfactory excision of the focus and then close the wound. The author's series represents every case of chronic bone infection that he has operated upon in the past two and one-half years,—and he has operated upon every patient who applied for treatment and did not refuse the operation. This series includes three patients who refused amputation. One of these is still disabled, one returned a year later and died of uraemia a few days after admission, and one has not returned. We are probably too slow in advising amputation in these old incurable infections of the bones of the lower extremities in adults.

The question arises, does the method of Dickson, Diveley, and Kiene for the treatment of chronic osteomyelitis have any advantage over the Orr method, which the author believes is the next best method? It does, because, in about 40 per cent. of the cases, the wounds may be closed and will heal by primary intention, if a satisfactory operation is performed. In adults, about 15 per cent. of the affected lower extremities should be amputated; the stumps may then be closed without drainage and will heal. In about 10 per cent., the patient will appear during an acute exacerbation of the infection; this can be relieved by sulfathiazole and drainage, and a radical operation which can be done later, if necessary. In the remaining 35 per cent. of the cases, most wounds may be almost

completely closed and will heal within a few weeks or months. There will be a few instances, probably 10 per cent., where amputation is not advisable and where healing does not follow surgery plus chemotherapy. We are anxious to know whether or nor penicillin will solve the problem presented by these.

It is probable that most of the cases which can be cured by the method of excision and primary closure can also be cured by the Orr method, but only after a longer interval and considerable unpleasantness. On the other hand, the closed method with chemotherapy is not dangerous and enables the surgeon to swing flaps, cover bones, and eliminate large areas of scarring in a manner which is not possible by any other method with which the author is familiar. The fact should be emphasized that this method is elastic and should be used in all cases of chronic pyogenic infection of bone, because a failure to obtain a complete success still represents an advance over older forms of treatment. This advance is measured by the degree of closure obtained and the consequent reduction in the area which must be covered by secondary healing. If acute infection is present and the bacteria are actively invading the tissues, the sulfathiazole should be used locally and systemically, and the focus should be drained. A radical operation with excision and closure can be done at a later date if necessary.

REFERENCES

Dickson, F. D.; Diveley, R. L.; and Kiene, Richard: The Use of Sulfathiazole in the Treatment of Subacute and Chronic Osteomyelitis. J. Bone and Joint Surg., XXIII, 516, July 1941.

Hoyt, W. A.; Davis, A. E.; and Van Buren, George: Acute Hematogenous Staphylococcic Osteomyelitis. Treatment with Sulfathiazole without Operation. J. Am. Med. Assn., CXVII, 2043, 1941.

DISCUSSION

Dr. H. W. Orr, Lincoln, Nebraska: Dr. Key says that the wounds in this War are being treated better because we are substituting sulfa drugs for Dakin's solution. Dr. Key has shown that patients have recovered after sulfa drugs by some such equation as this: an infected wound plus drainage plus sulfa drugs plus something else—and you get a recovery. What Dr. Key has shown this morning, and more nearly so than ever before, is what the patient does toward his own recovery. He has apparently not observed that this same program, with the sulfa drugs left out, will give us a high percentage of recoveries even if they take longer. It has been my feeling for a long time that, while a percentage of wounds may be closed primarily, another large percentage cannot be closed primarily and safely without risk to the patient. Many patients have recovered without credit to the doctor. The patient often has something in his system that allows closure without infection. What the sulfa drugs have to do with it, we do not know. We may continue to hope that some drug will be found sometime that will kill the staphylococcus without harm to the patient.

One of the questions I was asked to answer is: What are we to do about the odor in soiled dressings? My answer is that soiled dressings, or dressings with a "stench", represent the same thing in a dressing that infection does in a wound. It reflects a certain degree of discredit upon the surgeon. It has been our policy for a long time to dress these wounds with rather voluminous sterile dressings and with padding under the cast because we depend upon the pins embedded in the cast for immobilization. If moisture leaks through the sterile portion of a dressing to any outward surface which is not sterile, you have a culture medium extending all the way from the surface down to the wound. This is a fine field for the development not only of staphylococci or other infection, but for saprophytes that produce odor in the dressings. Prevention of contamination makes for prevention of infection and odor. If we prepare the wound and the cast so that no secretion exudes to the outside, odor will be prevented. That cannot be done at all times, but with a sufficiently sterile dressing, and with adequate disinfection chemically at the time of operation, the patient can be protected against that sort of thing. If complications occur in a wound, the surgeon will know how to protect the wound. If complications do occur, we may use heat about the wound area, just as we use heat for the wound itself.

Dr. Frank D. Dickson, Kansas City, Missouri: I am particularly pleased to discuss Dr. Key's paper, because our own interest in the local use of sulfonamides in osteomyelitis was stimulated by one of Dr. Key's original communications on the local use of these drugs. I would like to discuss this paper in an abstract way, avoiding controversial comparisons.

Dr. Key called attention to the fact that, if sulfonamides are going to be of value in local infection, three factors are important: first, the concentration of the drug in the immediate environment of the bacteria to be acted upon; second, the degree of concentration of sulfonamide-inhibitor substances present; and third, the effectiveness of the natural defense mechanism of the body. All investigation seems to indicate that a

higher degree of concentration of the drug in the immediate environment of the bacteria is secured by the use of the sulfonamides locally than can be obtained by any known safe method of systemic administration. This strongly suggests that, if the sulfonamides have any value, their local use is the most effective method of utilizing them. However, it is equally true that no matter how high a concentration of the drug is present in the environment of the bacteria, unless the introduction of the sulfonamides has been preceded by a thorough surgical clean-up of both bone and soft parts to eliminate or reduce to a minimum the sulfonamide-inhibitor substances (pus and necrotic tissue), you are not going to get results, since their action will be nullified. In other words, the primary factor of success is adequate surgery.

What does an adequate clean-up do when you are dealing with an osteomyelitic focus? It should do two things. First, it should reduce to a minimum the sulfonamide-inhibitor substances by removal of the source of these substances, which is pus and debris in the wound. Second, it should mechanically remove large quantities of bacteria. When these two purposes have been accomplished, the sulfonamides should be able to take care of what remains in the way of infecting bacteria, by inhibiting their growth, and to allow healing to take place by normal tissue repair.

As to primary closure, I am glad Dr. Key emphasized this fact, which we tried to emphasize when we originally reported our cases. If you are going to close a wound primarily, you must dissect away all scar tissue so as to bring the denuded bone into contact with healthy tissue with a good blood supply. I am convinced that bringing sclerotic scar tissue into contact with denuded bone is unphysiological. Certainly, its blood supply is poor and its lack of plasticity does not permit it to lie in close contact with the bone and so eliminate dead space, which is one of the primary factors in securing healing. So Dr. Key was quite right when he emphasized that this method of treatment was not advisable unless you could bring about a satisfactory closure. In about 80 per cent. of our cases of subacute and chronic osteomyelitis, in which the sulfonamides were used locally and systemically and primary closure was carried out, primary healing took place within three to four weeks. I believe our failures in the other 20 per cent. have been due to a poor selection of cases,—that is, we selected cases in which we were not able to get the proper tissue to cover the denuded bone. In but three or four of the failures has there been any extensive breaking down. There is usually a small area which does not heal, suggesting the existence of a dead space. If this method has any advantages, and we feel it has, there are two. In the first place, it gives rapid healing in three to four weeks. In the second, healing occurs with a minimum of scar-tissue formation. This is a very definite advantage if future reconstructive work is to be carried on. We have found it particularly useful in infected compound fractures, since the absence of extensive scar tissue enables us safely to apply a bone graft in eight to twelve weeks following healing.

Dr. J. Albert Key, St. Louis, Missouri (closing): I did not realize that I had given the impression that the patient did not have something to do with healing. Sulfonamides do nothing but inhibit the growth of bacteria. By so doing, the wounds can be closed with a good margin of safety, 99+ per cent. In 101 cases we had only one abscess that required opening. However, some wounds could not be closed completely and from others there was drainage between the sutures.

I think that Dr. Dickson is wrong when he says that a meticulous operation is necessary. On several occasions where there were deep abscesses extending up beneath the fascia, I simply curetted them, wiped them out with gauze, removed most of the infection, put in sulfanilamide powder, and immobilized the limb under a pressure dressing in a plaster cast. We used this method in every case, but were not always able to do a complete operation. It is a great advance over what we were able to do before the advent of the sulfonamides.

With his method, Dr. Orr may be able to keep the wounds dry, but I cannot. I do not like pin fixation with the Orr treatment, because pus may develop around the pin, and sometimes this leads to a diffuse infection of the bone.

CAPSULECTOMY FOR THE RELIEF OF FLEXION CONTRACTURES OF THE ELBOW FOLLOWING FRACTURE *

BY PHILIP D. WILSON, M.D., NEW YORK, N. Y.

Limitation of extension of the elbow is a not uncommon sequel of fractures in that region. The degree of the limitation varies considerably. Slight limitation — that is, less than 30 degrees — is not an appreciable handicap; and many adults, who have this amount of restriction of movement following fractures sustained in childhood, are scarcely conscious of anything wrong with their arms. The term flexion contracture is generally reserved to describe limitation of motion more serious than this, and it is to cases with extension limited at 120 degrees or less that this article applies.

If we may exclude the type of contracture caused by superficial scars, such as follow burns or lacerations of the soft parts, flexion contractures of the elbow resulting from fractures in that region may be due to one of the following conditions:

1. Irregularity or distortion of the joint surfaces, with loss of cartilage, or with fibrous adhesion;
2. Malunited fractures with bony projections or excessive callus forming a bony obstacle which impinges against the other member of the joint and impedes motion;
3. Fibrous thickening and contracture of the capsular ligament, causing loss of extensibility.

It is to emphasize the importance of this last factor as a cause of restricted motion of the elbow that this article is written.

There are, of course, many lesions of the elbow or adjacent soft structures, other than fractures, that may cause limitation of elbow motion. These include tuberculous, infectious, or rheumatoid arthritis, tumors, and various types of neurogenic contracture; but, since the treatment of these conditions is well understood, they will not be considered here, and the scope of the subject will be restricted to flexion contractures resulting from fractures.

PERIOD OF FUNCTIONAL IMPROVEMENT

It is first necessary to emphasize that limitation of extension of the elbow should not be designated as a flexion contracture until sufficient time has passed following the fracture to obtain restoration of function, if this is to occur. It is well known that recovery of motion following a fracture or dislocation of the elbow is often slow, and that gradual improvement is to be expected over many months. During these early months, when an anxious parent is complaining that the child's elbow is crooked and not getting better, the surgeon must calm these fears and counsel patience. Nothing but harm is done by forcible manipulation either with or without an anaesthetic. Physiotherapy is rarely indicated in the fractures of childhood, and is of only limited usefulness in those of adults. Active use of the arm within the limits of pain is the sovereign remedy, and a surgeon can usually accomplish more by encouraging the patient and counselling patience than by any other means. Skillfully supervised occupational therapy may be of considerable therapeutic assistance. Generally the greater part of the motion is recovered within six months, but a further gain of 10 to 15 degrees in the next six months is frequently observed. One is generally safe in assuming that functional recovery is complete at the end of one year, and that whatever limitation of motion is present at that time will be permanent.

DIAGNOSIS OF CAUSES OF FLEXION CONTRACTURES

Every case presenting limitation of motion following fracture of the elbow must be judged on its individual merits, and an attempt made to analyze the cause of the restriction

* Read at the Annual Meeting of The American Orthopaedic Association, Cleveland, Ohio, June 7, 1943.

of motion on the basis of the clinical examination and the roentgenographic evidence. Much may be learned by palpation and identification of the various bony prominences, and by ascertaining their relationship to each other. Also valuable information may be obtained by careful and repeated trials of the movement in an endeavor to determine whether it is limited abruptly, as by a bony obstacle, or whether the stoppage is of the less sudden and more elastic type characteristic of a contracted capsular ligament. When the limitation is present in both flexion and extension, it is generally a sign of intra-articular damage, and of involvement of one of the cartilaginous surfaces of the joint. Limitation of pronation and supination is also a sign of intra-articular damage, and generally means a malunited fracture of the head or neck of the radius.

The evidence obtained from roentgenographic examination is of great importance and the roentgenograms should be studied carefully. Narrowing of the joint cartilage space, or irregularity and distortion of one of the articular surfaces, points to intra-articular involvement as the cause of the limited motion. Bony irregularities and projections near the joint surfaces may mean a bone block to movement. The part that such bony obstacles play in impeding movement may be elucidated further by roentgenograms made with the elbow in complete extension and complete flexion, or by stereoscopic roentgenograms. It is from an understanding of the pathological changes which have taken place that the surgeon may make a diagnosis of the cause of the limitation of movement and determine what, if any, operative procedure may be undertaken to relieve the condition.

PATHOLOGICAL CONSIDERATION OF VARIOUS FRACTURES IN RELATION TO LIMITATION OF MOTION

1. *Supracondylar Fractures*

Here the fracture line is quite often intra-articular, entering the joint through the capsular prolongations at the coronoid or olecranon fossa, but there is no involvement of the articular surfaces. Commonly, the lower fragment is displaced posteriorly, although in rare cases, in the so-called "flexion type of fracture", it may lie anteriorly. There is hemorrhage in and around the joint, which later becomes absorbed or in part organized into fibrous tissue. The more perfectly the fracture is reduced, the less the amount of fibrous tissue that is formed. When union takes place with the lower fragment displaced posteriorly, there is increased opportunity for the accumulation of blood clot under the projecting end of the upper fragment, and this in turn is changed into fibrous tissue and may involve the anterior capsule. The position of flexion in which the elbow is usually fixed following reduction favors contracture and shortening of the anterior capsule. In the majority of cases, this contracture will gradually stretch and return to normal under the influence of constant active use. In a few cases, however, the fibrotic condition becomes fixed and a permanent contracture remains. In this type of case, if there is considerable limitation of motion, resection of the anterior capsular ligament and of the fibrotic tissue will lead to recovery of motion.

2. *Intercondylar Fractures*

These fractures involve the articular surfaces of the humerus, and generally cause considerable distortion or irregularity. The articular incongruity may of itself cause limitation of motion, and the uneven distribution of pressure may in time lead to additional erosion of cartilage with considerable osteo-arthritic reaction. Limitation of motion in this type of case may be partly due to some degree of fibrous ankylosis. The intra-articular changes are always associated with considerable periarticular fibrosis, which is also a factor in limiting motion. These fibrotic changes involve all parts of the joint capsule, and it is impossible to say that one portion is predominantly affected. Arthroplasty or excision of the lower end of the humerus is more likely to be successful in restoring motion than are operations to resect fibrotic portions of the joint capsular ligament.

3. *Fractures of the Olecranon*

Fracture of the olecranon is also an intra-articular fracture, but it is associated with a transverse tear extending through the lateral expansions of the triceps tendon and the posterior capsular ligament. When the fracture is treated in the modern manner by open reduction, strong internal fixation, and early mobilization, the chance of ensuing limitation of motion, either from joint irregularity or from fibrotic changes in the capsule, is small. When the fracture is comminuted, resection of the multiple fragments is generally the best treatment, and leads to little or no limitation of motion, because the movement can be started early before fibrotic changes develop. There are a few complicated fractures of the olecranon, usually associated either with anterior dislocation of the elbow or with fractures of the head and neck of the radius, where it may be necessary to fix the elbow in extension for several weeks. In such cases, fibrotic fixation of the posterior capsule and overlying expansions of the triceps muscle may make the recovery of motion slow and difficult. The author encountered one case of this type (Case 7) in which operation was required to restore flexion. After transverse division of the posterior capsule, it was possible to flex the elbow to a right angle and to allow mobilization following fixation for one week in plaster. The restoration of motion in this case was prompt and satisfactory.

4. *Fractures of the Head and Neck of the Radius*

Limitation of extension as well as of pronation and supination is common following fractures of the upper end of the radius, especially in the comminuted types. Excision of the radial head or of the multiple small fragments gives better results as far as functional recovery is concerned, when done early rather than late. When a poor result is obtained following such an operation, the limitation of motion is generally caused by an ossifying myositis or by calcification developing in the joint capsule. The failure to restore motion after late resection of the radial head (from six months to a year after injury) is, in the author's judgment, due chiefly to a lack of appreciation of the fact that the capsular ligament is extensively involved and that it plays an important rôle in causing limitation of motion. In the comminuted fractures, small fragments of bone are frequently driven anteriorly into or through the capsular ligament. When resection is done, the surgeon frequently fails to obtain sufficient exposure to visualize the lateral and anterior portion of the capsule. Exploration here frequently reveals the presence of one or more small bone fragments imbedded in a mass of scar tissue, effectively limiting not only extension but flexion of the elbow as well. When pains are taken to dissect out and remove this osteofibrotic mass, together with the deformed radial head, the results of late excisions are vastly improved. It cannot be emphasized too strongly that scarring of part of the capsular ligament plays almost as large a part in causing limitation of motion as does a displaced bone fragment.

ANTERIOR CAPSULECTOMY

The author's interest in this subject was aroused in 1932 when a girl of thirteen was referred to him because of limited motion of the elbow. The details of the history are as follows:

Case 1. L. R., a girl, aged thirteen years, was admitted to the Robert Brigham Hospital July 10, 1932. Four years previously she had sustained a supracondylar fracture which had been complicated by the development of Volkmann's ischaemic contracture of the hand and wrist. Several efforts at reduction had been made and union had taken place without satisfactory alignment being obtained. The elbow had been immobilized for about three months.

Examination showed a typical Volkmann's contracture of the hand and wrist. The fingers were fixed in marked flexion when the wrist was extended to the straight line, but they could be extended when the wrist was flexed. There was marked atrophy of the muscles of the forearm. The elbow showed marked limitation of motion with flexion to an angle of 65 degrees, extension to an angle of 80 degrees, and an actual range of motion of only 15 degrees. Supination was possible to a point halfway between full supination and the

FIG. 1-A

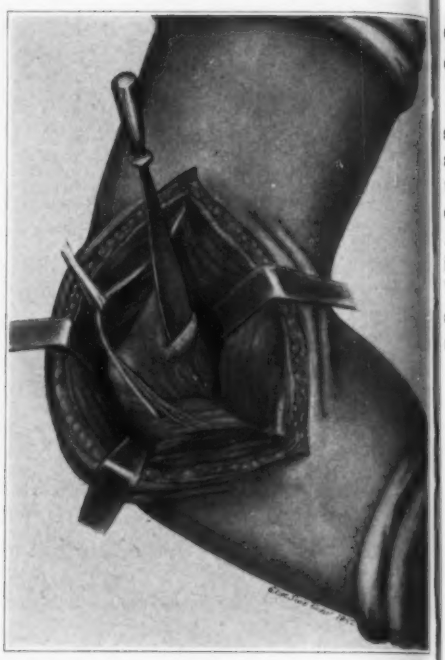


FIG. 1-B

Fig. 1-A showing the anterior approach to the elbow, the incision passing down between the brachioradialis and the brachialis, with exposure and retraction of the radial nerve.

Fig. 1-B showing exposure of the lower end of the humerus by subperiosteal stripping, followed by chiselling away of the bony irregularity on the outer surface of the humerus just above the capsular attachment.

FIG. 1-C

The bone fragment, scar tissue, and anterior capsule have been stripped downward preliminary to excision of these tissues.

mid-position, and pronation was limited at a point slightly beyond the mid-position. The roentgenographic examination showed an old malunited supracondylar fracture, with posterior displacement of the lower fragment and an anterior bony projection which evidently represented the lower end of the upper fragment. The articular cartilage space at the elbow appeared normal.

Operation

It was necessary to do something to restore motion at the elbow. The author thought that the thickened capsule was playing a considerable part in limiting motion, and advised an operation to explore and remove the contracted portion. The operation was performed on July 11, 1932. An anterior incision was made between the brachioradialis and the brachialis. The radial nerve was exposed and retracted. The anterior capsule of the elbow was found to be greatly thickened, with fibrous bands passing down into it from the space below the bony projection on the anterior surface of the shaft just above the joint. This bony projection was excised, together with as much of the anterior capsular ligament as could be reached. A Z-lengthening of the biceps tendon was done. Extensive bleeding during the operation necessitated the application of an axillary tourniquet. The removal of this mass of scar tissue allowed the elbow to be extended immediately, but placed the radial nerve under a great deal of tension. After closure of the incision, a circular plaster

casing was applied from the wrist to the axilla, with the elbow fixed at an angle of 120 degrees of extension.

In this case the postoperative course was complicated by a hematoma, and there were characteristic signs of a complete radial-nerve palsy, with wrist-drop and loss of extension of the thumb, but it was deemed advisable to continue the stretching of the elbow notwithstanding. During the second week, the patient was placed in traction with an overhead frame. After sixteen days, the plaster was cut transversely at the elbow, and a turnbuckle was installed, which was lengthened every day to give increased extension. By August 7, 1932, the elbow had been extended to an angle of 150 degrees, and the patient was discharged wearing a removable plaster splint which was to be used chiefly at night. She then returned to her home in Wisconsin, and the after-care was under the direction of L. D. Smith, M.D., of Milwaukee, who, in 1937, sent the author an end-result report. His note described the patient on March 2, 1937, as "fairly active athletically, adjusted, and well compensated. The angle of flexion of the elbow was 62 degrees, extension

FIG. 2-A

Case 2. J. U. November 29, 1941. Roentgenographic appearance of the elbow in extension and flexion seven years after operation.

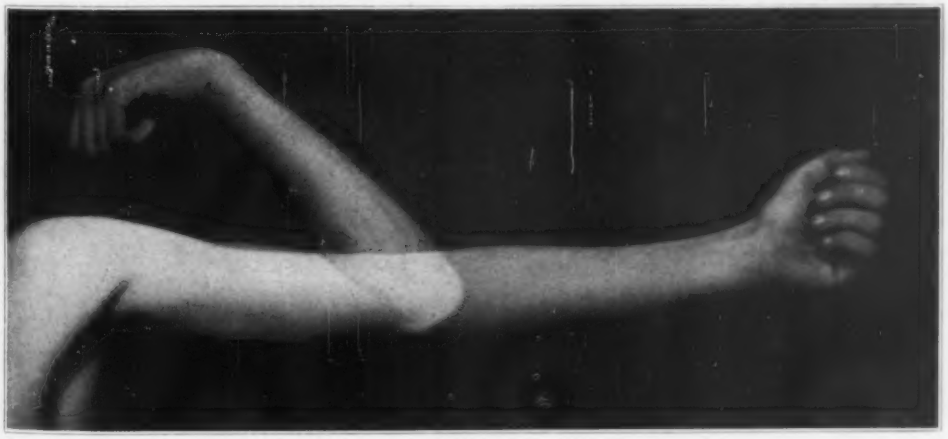


FIG. 2-B

Photographs taken December 6, 1941, showing elbow function.

was possible to an angle of 146 degrees, wrist flexion to 130 degrees, and wrist extension to 162 degrees. Pronation and supination were not recorded.

This case demonstrated beyond doubt that fibrous thickening of the anterior capsule may play a considerable part in causing limitation of elbow extension, and that the condition may be overcome by excision of the fibrotic portion of the capsular ligament. While the cases in which such procedures may be required are rare, the author has accumulated five in a period of eleven years, and feels that it may be worth while to report his experience in the hope that it may be of help to other surgeons.

The operation has generally been performed under a tourniquet through an anterior incision passing between the brachioradialis and the brachialis. This incision gives good visualization of the contracted capsule, and, at the same time, allows identification and freeing of the radial nerve. It has the disadvantage of crossing the flexion creases of the skin at the anterior surface of the elbow, and in children tends to give rise to keloid formation. In the last operation which was done, a medial incision was used, the muscles being stripped subperiosteally from the anterior surface of the humerus. This gave adequate exposure of the anterior capsule, without the disadvantage of cutting the anterior skin creases. When another patient requiring this operation is encountered, the medial incision will be used.

The record of complicating nerve palsies following operation has been bad, occurring in four out of five of the cases. In two of these patients, the palsy was caused by stretching the radial nerve which had become contracted because of the long-continued position of flexion; in the other two cases, it was probably the result of pressure from the tourniquet. As desirable as a bloodless field may be for this type of operation, it is not worth the risk of this complication, and it would be better to discard the use of the tourniquet. Complete spontaneous recovery of all these palsies was obtained within a period of four months, so that no permanent residual damage resulted from any of the operations. Whether this was due to good luck or good management does not matter, but profit should be drawn from this experience for the future.

FIG. 3-A

Case 3. A. A. August 17, 1937. Photograph showing elbow extended to maximum at the time of neurolysis of the radial nerve for radial palsy.

FIG. 3-B

October 30, 1939. Roentgenographic appearance of the elbow just prior to anterior capsulectomy.

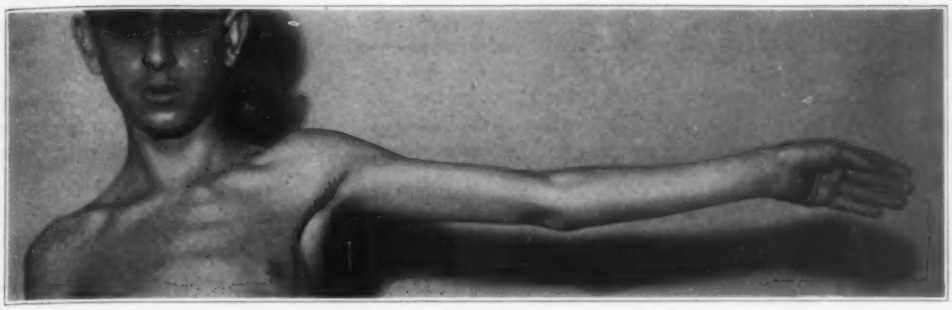


FIG. 3-C

May 21, 1941. Photographs show the elbow function nineteen months after operation.

REPORTS OF OTHER CASES

CASE 1. L. R. (See pages 73, 74, 75).

CASE 2. J. U., a boy, aged seven and one-half years, was first admitted to the Hospital for Special Surgery May 17, 1933. He had sustained a supracondylar fracture of the left humerus February 10, 1933. At the time of admission, the range of motion of the elbow was from 85 to 115 degrees. An osteotomy was done May 25, 1933, to correct the bony deformity, and a projecting fragment of the bone was removed. The elbow was immobilized in complete flexion for about four weeks. Following the removal of the plaster cast, progress was not satisfactory, and he failed to recover extension of the elbow.

On admission September 18, 1934, examination showed elbow flexion to be 75 degrees, extension 120 degrees; and pronation and supination to be complete.

He was operated upon September 20, 1934. Dense scar tissue in front of the elbow, attached to the humerus at the fracture site and to the anterior capsule, was excised, and the projecting bone was removed.

FIG. 3-D

December 6, 1941. Roentgenographic appearance approximately two years after the operation, showing the maximum flexion and extension. Note the reduction in the size of the anterior bony projection.

There was flexion to 30 degrees and extension to 150 degrees. The elbow was placed in a plaster cast in extension of 150 degrees. The cast was removed after one week, and active motion was begun. There were no complications.

Seven years later, November 29, 1941, the elbow had complete extension, and lacked but 5 degrees of complete flexion (Figs. 2-A and 2-B). Pronation and supination were complete. There was a scar at the front of the elbow which was rather tight on complete extension, but it did not cause any limitation of extension or any functional difficulty.

CASE 3. A. A., a boy, aged ten years, was first admitted to the Hospital for Special Surgery August 16, 1937. He had sustained a supracondylar fracture of the left humerus May 1, 1937, and had been treated in a plaster cast for eighteen days. At the time of admission he had motion in the elbow of from 60 to 90 degrees. He had a complete paralysis of the radial nerve, both sensory and motor. On August 18, 1937, a neurolysis was done. Following this, there was a rapid return of sensation; motor power began to return in three weeks, and continued to improve until January 22, 1938, when motion of the elbow was from 50 to 95 degrees. There was no further improvement during a period of two years, and operative treatment was advised.

FIG. 4-A

Case 4. A. W. Roentgenograms showing the characteristic supracondylar fracture sustained on July 25, 1938.

FIG. 4-B

July 6, 1939. Roentgenograms showing the appearance of the healed fracture one year later, and the angle to which extension was limited.

On admission, examination revealed flexion in the elbow to an angle of 50 degrees, extension to an angle of 95 degrees, supination limited by 15 degrees, and pronation limited by 5 degrees. Roentgenograms showed a healed supracondylar fracture of the humerus, with prominence of the distal end of the proximal fragment anteriorly.

The patient was operated upon November 3, 1939. With a tourniquet in place, the front of the elbow was exposed through an incision passing between the brachialis and the brachioradialis. The projecting bone was excised with an osteotome, and the scarred anterior capsule was completely removed by sharp dissection. A plaster cast was used for immobilization.

The cast was removed seventeen days after the operation, and physiotherapy and active motion were begun. A complication of incomplete paralysis, involving the median and radial nerves ended in complete recovery after about four months. By December 6, 1941, the patient had normal use of the elbow, with flexion to 35 degrees, extension to 170 degrees, pronation limited to 15 degrees, and supination complete. There was no residual nerve paralysis.

FIG. 4-C

November 16, 1941. Photographs showing the range of elbow function twenty-eight months after operation.

CASE 4. A. W., a girl, aged six and one-half years, was admitted to the Hospital for Special Surgery July 5, 1939. On July 25, 1938, she had sustained a supracondylar fracture of the right humerus with the lower fragment completely displaced backward and rotated about 60 degrees. Satisfactory reduction had been obtained the same day, and had been held in place with a bandage. On August 11, 1938, roentgenograms had shown a recurrence of the displacement, and the arm had been remanipulated and placed in a cast. The cast had been removed in six weeks. Four months after injury, a turnbuckle cast had been applied to increase the range of extension, but little improvement had been obtained.

On admission, the elbow flexed to 60 degrees, and extended to 110 degrees; pronation lacked 20 degrees, and supination was normal. Roentgenograms revealed an old supracondylar fracture, with a beak of bone on the proximal fragment projecting anteriorly.

She was operated upon July 7, 1939. Excision of the bony spur and of the scarred anterior capsule at the elbow was done. The elbow was placed in a plaster cast, with extension to 140 degrees. Radial palsy was again a resulting complication, but cleared up after four weeks.

Photographs taken November 16, 1941, showed that elbow motion was from about 40 degrees to 180 degrees. Pronation and supination were not shown. The mother reported the arm to be "about perfect".

CASE 5. I. L., a boy, aged twelve years, was first admitted to the Hospital for Special Surgery October 16, 1939. He had sustained a supracondylar fracture of the left humerus twenty-two days previously. Two attempts had been made at reduction. A board splint had been applied, apparently with the elbow in extension. Later, he had been transferred to this Hospital.

On admission, examination showed the elbow to be markedly swollen and discolored. It was fixed at an angle of 150 degrees of extension. There was only 3 or 4 degrees of motion. Pronation was limited 30 per cent., while supination was complete. Sensation was

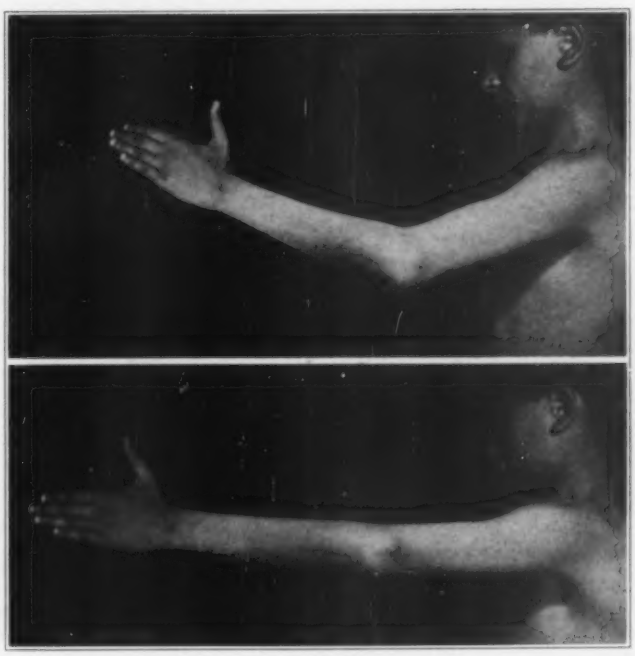


FIG. 5-A

Case 5. I. L. July 16, 1940. Position and range of motion before operation.

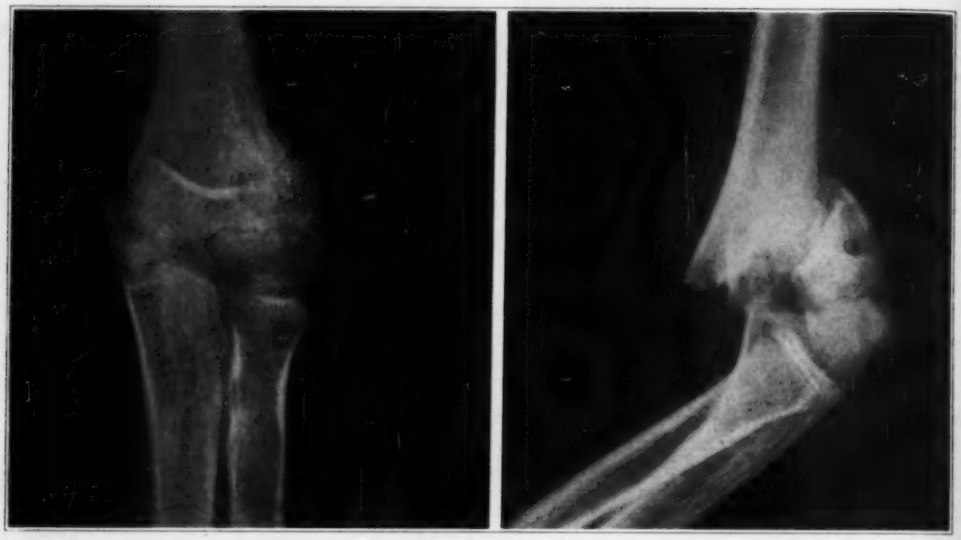


FIG. 5-B

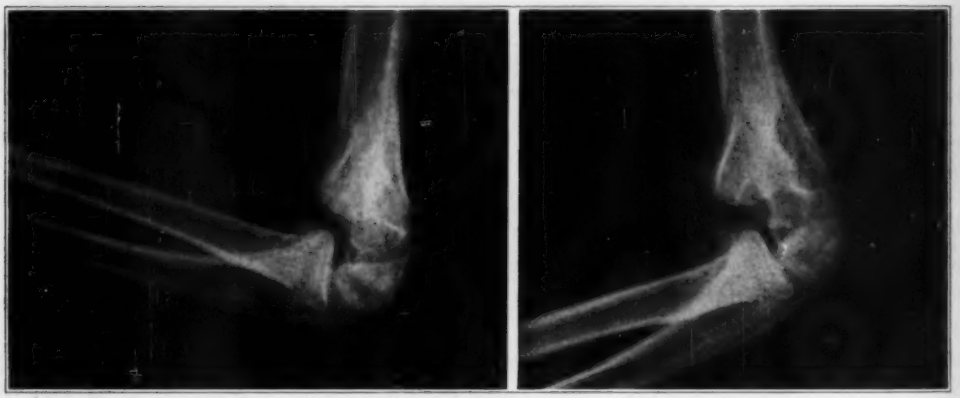


FIG. 5-C

FIG. 5-D

Fig. 5-B: October 16, 1939. Roentgenogram of the original fracture.

Fig. 5-C: August 17, 1940. Appearance of elbow immediately following the first operation. There still appears to be considerable bony prominence at the lower end of the humerus just above the joint.

FIG. 5-D: June 17, 1942. Appearance and range of motion of elbow prior to the second operation.

normal, and there was no circulatory impairment. Roentgenograms showed a supracondylar fracture, with the lower fragment displaced backward nearly 100 per cent., and tilted backward 25 degrees. There was a moderate amount of fairly dense callus.

It was not considered advisable to attempt further improvement in the position of the fragments. Traction was applied to the forearm for two weeks without changing the position of the fragments. A plaster was then applied with the elbow in as much flexion as possible. Active motion was begun after another ten days.

The patient was discharged November 18, 1939, without support. He was followed in the Out-Patient Department, but showed very little improvement in range of motion.

On July 15, 1940, he was readmitted to the Hospital. At that time the elbow could be extended to an angle of 160 degrees, and flexed to an angle of 115 degrees. Supination was normal, but pronation was limited to 70 per cent. Roentgenograms showed that the fracture was united, but with posterior displacement of the lower fragment.

The patient was operated upon July 19, 1940. The elbow was approached through an anterolateral incision between the brachioradialis and the brachialis. A large projecting mass of bone representing the lower end of the old upper fragment was removed with an osteotome, together with a mass of fibrous tissue involving the anterior capsule. With moderate force, the elbow could be flexed to an angle of 60 degrees. After closure, the elbow was immobilized in a plaster cast, in flexion a little beyond a right angle.

The cast was removed after seven days. The arm was then put in a sling in acute flexion until the tenth day after operation when active physiotherapy was started. Cooperation was considered poor. The patient was discharged August 24, 1940, with extension to 120 degrees, flexion to 70 degrees, and with supination and

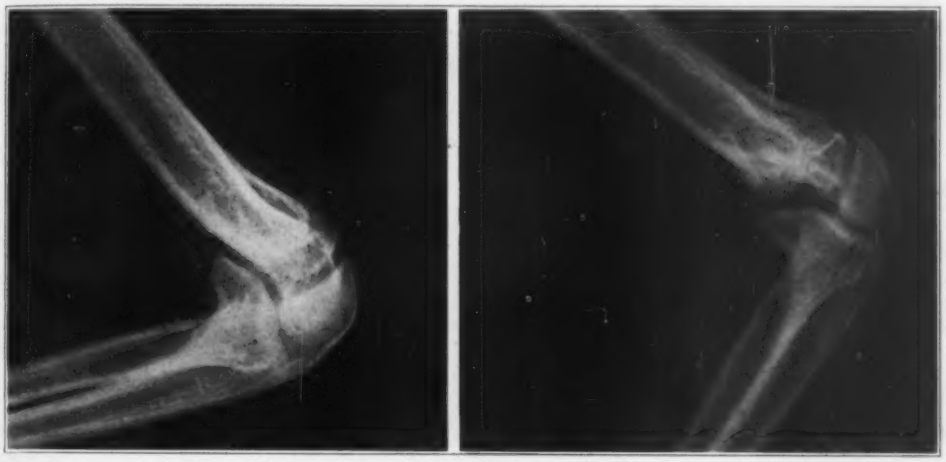


FIG. 5-E

June 17, 1942. Roentgenograms of the elbow in maximum flexion and extension prior to the second operation.

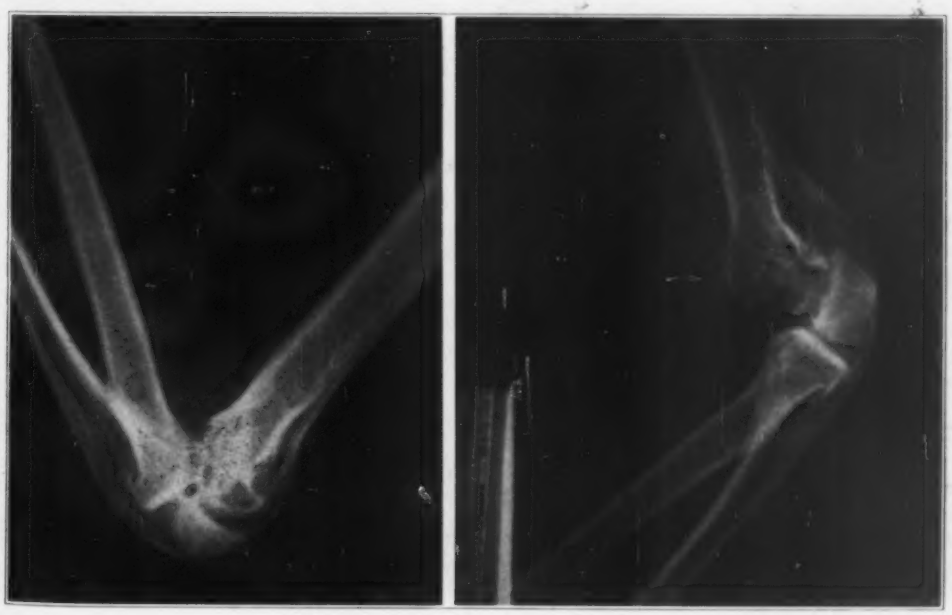


FIG. 5-F

November 30, 1942. Five months ofter operation some improvement in motion has been obtained and more may be expected.

FIG. 6-A

Case 6. C. K. August 22, 1936. Roentgenogram of elbow prior to excision of the radial head.

FIG. 6-B

September 18, 1936. Roentgenogram showing the immediate postoperative result.

pronation complete. He was followed in the Out-Patient Clinic. Radial nerve paralysis was recorded December 21, 1940, but was followed by complete recovery. In January 1942, the range of motion was from 60 degrees flexion to 90 degrees extension, and he was, therefore, advised to come in again for another operation.

On June 17, 1942, he was admitted for the third time. Examination showed a range of motion from 55 degrees of flexion to 90 degrees of extension. Supination was complete, while pronation was limited 15 per cent.

The patient was operated upon June 20, 1942. The elbow was exposed by two incisions, one on the medial side, and one on the lateral side. Many dense scar bands across the anterior capsule were found, and dissected free with the capsule. The brachialis tendon was sectioned. After closure, the arm was put in a plaster cast in as much extension as possible.

The cast was removed after ten days, and active motion was started. The patient was more cooperative in treatment and showed considerable improvement following discharge.

On January 5, 1943, a range of motion from 75 degrees of flexion to 135 degrees of extension was present. Pronation and supination were complete.

CASE 6. C. K., a male, aged twenty-eight years, was admitted to the Hospital for Special Surgery October 14, 1938. He had been seen first September 30, 1938, when he gave the following history: In June 193[illegible]

he had fallen eight feet, striking the left elbow, and sustaining a dislocation of the elbow and fracture of the head of the radius. The dislocation had been reduced within one hour. The following day, an operation had been performed on the elbow to remove a "broken fragment of bone". Movement had remained limited, and in September 1936, a second operation had been performed to remove the head of the radius. Upon admission, he complained of stiffness and aching pain in the elbow.

Examination showed no bone deformity, but marked restriction of motion. Flexion was possible to an angle of 60 degrees, and extension to an angle of 100 degrees, giving an arc of motion of 40 degrees. Rotation of the forearm was also limited, with supination of about 25 degrees beyond the mid-position and pronation of about 15 degrees. Function of the hand, wrist, and shoulder was normal.

A review of the various roentgenograms, going back to the time of the original injury, showed a comminuted fracture of the head and neck of the radius with a large fragment representing part of the circumference of the head displaced posteriorly and medially. At the first operation, this fragment had been re-

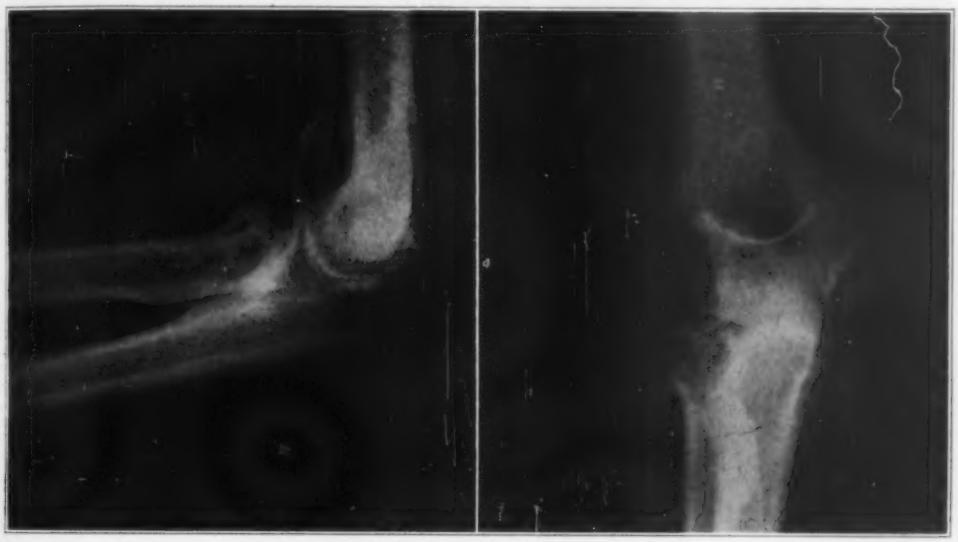


FIG. 6-C

October 14, 1938. Roentgenogram of elbow prior to capsulectomy. Flexion is possible to an angle of 60 degrees, extension to an angle of 100 degrees. Only a few degrees of rotation of the forearm is present.

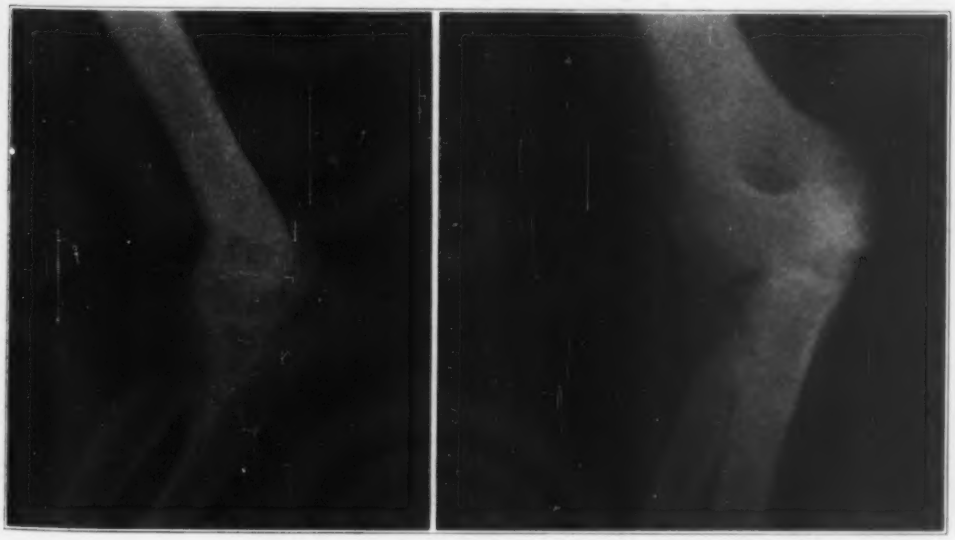


FIG. 6-D

October 28, 1939. One year after the operation, flexion is possible to an angle of 60 degrees, and extension to 140 degrees; pronation and supination are complete.

moved. At the second operation, a partial resection of the radial head had been done. Roentgenographic examination in October 1938 showed an old deformity of the radial head, suggesting partial removal, and a good deal of bone proliferation. Large deposits of bone were seen in the radial head laterally. There was a large bony mass arising from the medial surface of the humerus just below the epicondyle, and, anterior to

FIG. 7-A FIG. 7-B

Fig. 7-A: Case 7. J. B. February 7, 1939. Showing fracture of the olecranon, with anterior dislocation, and fracture of the humerus in the middle third. The elbow was treated by open reduction and was fixed in extension.

Fig. 7-B: May 26, 1939. This treatment resulted in a stiff elbow.

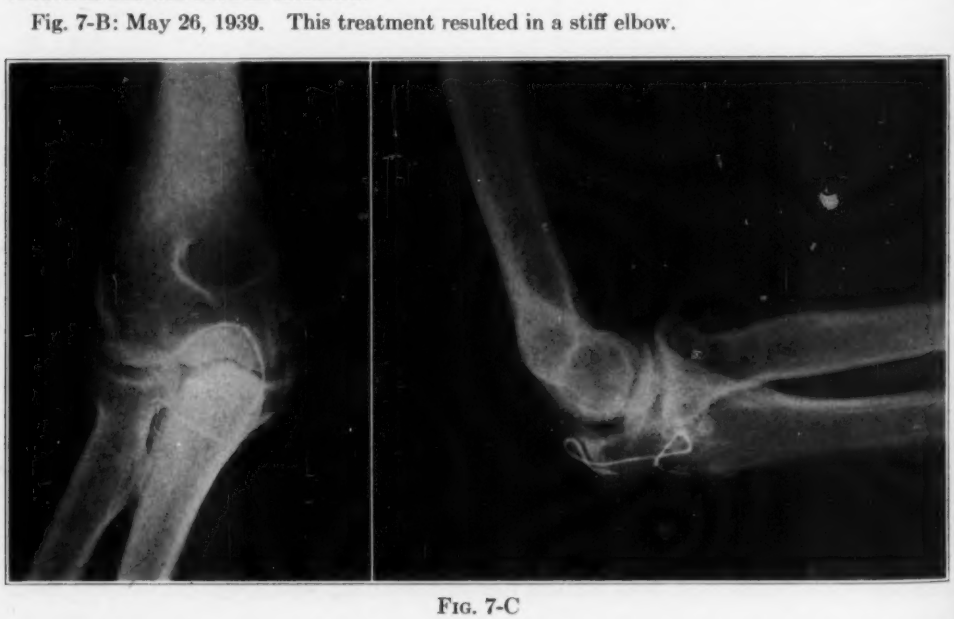


FIG. 7-C

June 8, 1940. Showing result one year after healing of fracture and posterior capsulectomy. Flexion is possible to an angle of 55 degrees, extension to 155 degrees; pronation and supination are complete.

the lower extremity of the humerus on a sagittal plane between the radius and the ulna, a dense curved arc of calcification suggesting myositis ossificans was found.

The patient was operated upon October 15, 1938. The front of the elbow was exposed through a long incision between the brachioradialis and the brachialis. A bony mass, two inches by three-quarters of an inch, was excised from the substance of the brachialis. The head and three-quarters of an inch of the shaft of the radius were removed. Through a posterior incision extending down from the lateral condyle, the ossified portion of the posterior capsule was excised. Through a third medial incision posterior to the internal epicondyle, the thickened anterior capsule and some more newly formed bone were removed. The elbow could be extended considerably beyond the preoperative limit. A Penrose drain was left in the posterior incision, extending into the antecubital space. It was removed after three days. The elbow was immobilized in a plaster cast in complete supination and 140 degrees of extension. Two weeks later the plaster was removed and active mobilization was begun. Progress was rapid and satisfactory, and there were no complications. The patient returned to work as an engineer in three months.

On November 26, 1941, he had full use of the arm and no complaints. The range of motion was flexion to an angle of 60 degrees, and extension to an angle of 140 degrees. There was complete supination and pronation. Roentgenographic examination showed that the upper end of the radius had been removed just above the bicipital tubercle, and that the calcified area previously visible at the front of the elbow was gone.

CASE 7. J. B., a male, aged eighteen years, was admitted to the Hospital for Special Surgery February 6, 1939. The patient had been injured two days previously when an automobile in which he had been riding side-swiped a snow plow and turned over. He had been treated at an adjacent hospital where roentgenograms had revealed fractures of the right humerus and elbow and of the right mandible. Plaster splints had been applied, and he had been transferred to this Hospital.

Examination showed marked swelling and ecchymosis about the elbow, and abnormal mobility at about the middle of the right humerus. The circulation in the hand was good, and there was no evidence of nerve involvement. Roentgenographic examination showed a fracture of the shaft of the right humerus in the middle third with angular deformity, fracture-dislocation of the right elbow, with extreme comminution and separation from the proximal fragment of about two inches. The ulna and radius were dislocated anteriorly on the lower end of the humerus. There was a fracture through the right ramus of the mandible.

On February 8, 1939, under ether anaesthesia, the fractured jaw was treated by wiring the mandible. Closed reduction was done on the fracture of the right humerus, and an unsuccessful attempt was made to reduce the dislocation of the elbow.

On February 17, 1939, open reduction for the fracture-dislocation of the elbow was carried out. The comminuted fragments of the olecranon were replaced and fixed with steel wire. A plaster spica, with an arm-piece extending down to the hand, was applied, fixing the elbow in complete extension.

Postoperative roentgenograms showed that the fracture of the humerus was in good alignment and approximation, and that the fracture-dislocation of the elbow had been reduced. The patient was up and about, walking in the plaster spica, when discharged. The spica was removed April 14, 1939, at which time there was solid union of the fractured humerus. Physiotherapy and efforts at active mobilization of the elbow were begun.

Examination May 17, 1939, showed good function in the hand, wrist, and shoulder. The right elbow was fixed at an angle of 150 degrees of extension, but only about 5 degrees of motion was possible. Supination was possible through about half the normal range and pronation was about half of normal. Roentgenographic examination showed solid union of the olecranon. The joint cartilage looked normal, and there was no calcification in the capsule. Operation to free the capsular adhesions was advised.

Operation was done May 19, 1939, through a mid-line incision over the posterior surface of the lower humerus and ulna. Definite fibrous thickening was encountered under the triceps tendon at the radial side of the olecranon, and a band of capsule was divided. A second incision through the capsule and triceps tendon was made on the medial border of the olecranon. The ulnar nerve was freed and retracted, and the capsular adhesions were divided. It was then possible to flex the elbow to above a right angle. The fracture itself was solidly healed. After closure of the incision, a plaster cast was applied, fixing the elbow in right-angle flexion.

One week after operation, the plaster was removed, and active mobilization was begun. Progress was satisfactory. November 29, 1941, the patient was using his arm quite normally. Extension was possible to an angle of 155 degrees, flexion to an angle of 55 degrees. Pronation and supination were complete. He had normal use of the hand and wrist.

SUMMARY

Pathological changes caused by fractures about the elbow may result in shortening and loss of extensibility of the anterior capsular ligament. By excision of the contracted and fibrosed portion of the anterior capsule, the author has improved the condition in five patients with flexion contracture of the elbow following supracondylar fractures.

A contracted fibrotic capsule may play a rôle in the limitation of motion in other types of fracture, and two cases are reported (Cases 6 and 7), showing that improvement of function may be obtained by operations designed to free the contracted portions of the capsular ligament posteriorly and laterally as well as anteriorly.

DISCUSSION

DR. EDWARD HARLAN WILSON, COLUMBUS, OHIO: In discussing this paper may I say that I am in agreement with the principles set forth therein. It so happens that I have had some experience with a similar procedure in three cases.

The first patient was a soldier who came under my care in England last year, while I was with the American Hospital in Britain. He had a flexion contracture following an inconsequential type of fracture of the humerus. We did an anterior capsulotomy very similar to that described in this paper. He was accepted back for service two weeks later.

Since then I have had two cases. One of these patients had been refused by the Navy because of a persistent flexion deformity of several years' standing following a fracture. Again, the fracture itself was not sufficient to block elbow motion. Capsulotomy was done, and four months later he was accepted in the Navy. In neither this nor the previous case did full extension follow operation, there being about 15 degrees of limitation. The striking thing to me was the rapidity with which function developed after use was begun.

In a third case, an old Volkmann's contracture, about 50-per-cent. improvement resulted from the same procedure.

Referring to the matter of the tourniquet, I would like to add that a pneumatic tourniquet dressing may be used on the arm without causing nerve paralysis, such as is reported in these cases, if it is interrupted every fifteen minutes.

DR. LEO MAYER, NEW YORK, N. Y.: I want to congratulate Dr. Wilson on his brilliant results. I wish he would tell us a little more about how he lengthened the biceps tendon and the tendon of the brachialis. I found that lengthening these structures caused me a great deal of trouble. When I straightened the arm the radial pulse would disappear, and then I would have to put the arm back in the original position of flexion. I would like to know how he overcame this difficulty.

DR. PHILIP D. WILSON, NEW YORK, N. Y. (closing): I agree with Dr. Harlan Wilson's critical comments about the nerve lesions, which I am sure followed the use of a tourniquet. I have experimented with various types of tourniquet and generally use an air-pressure cuff, but the trouble with this method is that in a small arm the cuff takes up too much room. The operation can be done without a tourniquet, but undoubtedly is much facilitated by its use. I think probably the best method, and the one I shall use in the future, is to employ a sterile tourniquet which I can put on and remove at half-hour intervals to prevent too much pressure on the nerve or too long ischaemia of the nerve. However, all the nerve complications in these cases cleared up and made no difference in the eventual result.

It is not necessary to completely extend the elbow at the time of operation, and I feel that the position of complete extension should be avoided when the plaster splints are applied. In this position, tension is placed on the contracted nerves and arteries and there is greater danger of some complication from their stretching. It is sufficient to fix the elbow in a comfortable position of extension for a period of approximately seven days and then to begin movement. I would rather depend on active exercise to recover full extension than on stretching the elbow under anaesthesia. In my first case, I used a turnbuckle plaster, but I think the result would have been just as good if we had removed the splints and relied on exercise after a period of seven to ten days.

THE KENNY CONCEPTS AND TREATMENT OF INFANTILE PARALYSIS

BY A. BRUCE GILL, M.D., PHILADELPHIA, PENNSYLVANIA

The Kenny concepts and methods of treatment of infantile paralysis are described as "world famous". They have been widely publicized in newspapers, magazines and journals, and over the radio. The influence of this publicity upon the public has been so great that the parents of children who have been afflicted with infantile paralysis are beginning to demand that their children be treated by the Kenny method. In other words, the layman is telling the doctor how he should treat a specific disease with its complications.

This dangerous publicity has not waited for a scientific or experimental examination of the claims and the merits of this method; it has not waited for a free and frank discussion by the medical profession; it has not waited even for sufficient clinical observations throughout the country to test the end results obtained by this method, which has been proclaimed as "unique", "original", "revolutionary", and "fundamentally different from that hitherto prevailing". To be guided by a publicity and methods of propaganda which outrun scientific research, clinical observations, and rational analysis and discussion is dangerous for physicians and for the public. Unless all the claims made for it are eventually substantiated, unless it proves to be a cure for infantile paralysis and its complications, or, at least, a very definite and apparent improvement upon the so-called "orthodox" methods, the public will have some justification for believing that it has been deluded, and physicians who have been swept away from their positions of scientific observation and rational criticism will have reason to feel some inward embarrassment and humiliation.

Physicians must maintain their independent scientific principles of investigation, discussion, and decision in the face of overwhelming popular demand and pressure by influential persons and organizations, and in spite of unpopularity or adverse criticism occasioned by their attitude. Whatever our final judgment of the claims made for the "Kenny method" may be, it must be based on medical knowledge and experience. That medical knowledge and experience are at present complete, no one will maintain. We seek to enlarge them, and welcome scientific investigation of the nature of disease and of its treatment.

Since Miss Kenny came to this country in 1940, she has been given ample opportunity to state her concepts and to demonstrate her methods of treatment. Many doctors have visited her clinic in Minneapolis, and in many clinics throughout the country her methods are being given trial. While some doctors have enthusiastically approved her concepts and methods and have endorsed them in print, the author believes that Miss Kenny is far from the truth when she states that they have been accepted by the American medical profession and by the American Medical Association (Pohl and Kenny, p. 25 and third page of jacket).

Because of the enormous publicity which has been given to Miss Kenny and her extraordinary claims, and in accordance with the constant desire of the medical profession to seek the truth concerning the cause, the nature, and the treatment of disease, a consideration and an analysis of the assertions made by her and her proponents are appropriate at this time.

The essential features of her treatment are stated to be the absence of immobilization and splinting, the meticulous use of hot packs, and a new system of muscle re-education. Since she does employ a method of immobilization which is the equivalent of splinting, since heat, both moist and dry, has been advocated and used heretofore, and since her principles and methods of muscle re-education are practically identical with those which

have long been known and taught as standard and "orthodox" (these statements will be proved later), there is scant basis for the use of the emphatic adjectives mentioned above.

However, Miss Kenny enunciates a concept of the nature, or pathology, of infantile paralysis which, it must be admitted, is unique. To one who tries to comprehend the exposition of this concept, it appears as an effort to mix some of the facts known to scientific medicine with strange and unproved theories and fancies. Looseness of statement, confusion, contradictions, and distortions are apparent, as will be pointed out in subsequent paragraphs.

The Kenny proponents summarize these revolutionary claims by stating that "orthodoxy has erred both in the recognition as well as the interpretation of the physical findings in the disease. . . .

"Orthodoxy has largely been devoted to treating normal muscles and has failed to recognize the true condition in the muscles affected by the disease. This then is the real contribution of Miss Kenny, not a new treatment of infantile paralysis as it has been conceived in the past but a conception of the disease itself so radically opposed to the old as to almost warrant considering the entity as a new disease. . . .

"The flaccid muscles are normal. Loss of ability to contract these is due to functional dissociation (alienation) from the nervous system.

"Ability to voluntarily contract the non-functioning muscles returns only after releasing spasm in the opponents and carefully restoring the physiological continuity of the nerve conduction paths back to the muscles.

"Paralysis due to nerve cell death occurs but it is not a common condition. Most supposed weakness is due to untreated spasm and to disuse in the dissociated muscles. . . .

"Deformities do not occur. Those resulting from the old methods were due to untreated muscle spasm.

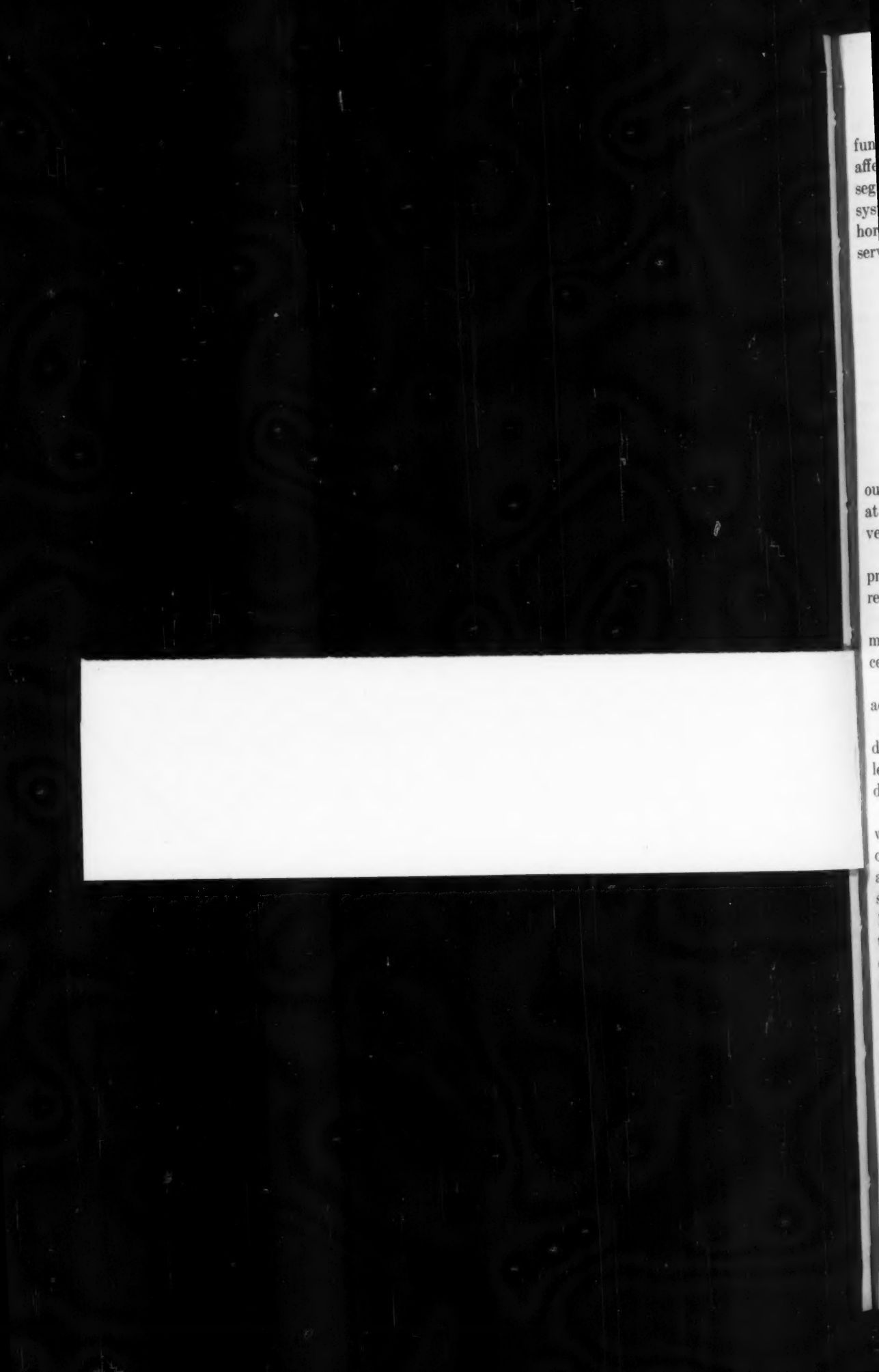
"In short, Miss Kenny's discovery is that infantile paralysis is a disease in which disturbance of physiological function of the nervous system is of more importance than actual architectural change. Many of the observable clinical phenomena are the result of functional disorganization of the motor centers and of the nerve pathways to the muscles. The disease affects muscle as well as nerve tissue. Muscle spasm is the primary lesion in the disease rather than paralysis. Miss Kenny has designed a treatment for these conditions. Needless to say, the treatment could have nothing in common with the previous methods, designed for a disease of opposite conception" (Pohl and Kenny, pp. 37, 38).

The validity of this last statement will be controverted, if it be demonstrated that the two methods of treatment have much in common, even though the two concepts are vastly different. Miss Kenny's claim that "orthodoxy" has failed to recognize the physical findings in this disease will be shown to be false. The proper interpretation of these symptoms will be discussed. That paralysis is due to some obscure disturbance of the physiological function of the nervous system and not to structural changes in the brain and the spinal cord will be shown to be without basis and opposed to incontrovertible evidence. There is no evidence to show that the virus of this disease affects muscles and peripheral nerves. Muscle spasm is a reflex condition, and is primarily due to the lesions in the central nervous system. Paralysis, whether complete or incomplete, whether permanent or temporary, is due to damage to the neurons. When the Kenny concept arrays itself against the vast accumulation of scientific evidence as to the nature of infantile paralysis, it cannot but go down in defeat. This is not to be construed to mean that our scientific knowledge of this disease is complete and that we do not welcome and search for additions to it, but new contributions must be made on a proved scientific basis.

Miss Kenny's claims to originality and her concepts and methods of treatment are based fundamentally on her observations of three symptoms, or physical findings,—"spasm", "alienation", and "incoordination", and on her deductions as to the nature and the importance of these symptoms.

Correction

The above statements which mention the pathology of the disease as found in the central nervous system are, of course, in accord with the standard teaching and are not a discovery of Miss Kenny, although no mention is made in the articles quoted that would lead the reader to believe that it was other than Miss Kenny's concept. However, their description of the proved pathology is not sufficiently complete.



"The concept of infantile paralysis on which Miss Kenny's treatment is based is fundamentally different from that hitherto prevailing. The disease in the acute stage affects not only the anterior horn cells but also adjacent portions of the cord. It may be segmented in character, involving the central nervous system including the sympathetic system in general so that symptoms other than those due to involvement of the anterior horn cells must be present. The symptoms, as pointed out by Miss Kenny and as observed by us, are mainly:

1. The muscles affected present the condition of spasm.
2. The affected muscles become shortened.
3. Coordination is disorganized and incoordination frequently seen.
4. The patient frequently loses power in non-affected muscles because affected muscles are pulling the non-affected muscles from their normal resting place and retaining them in their lengthened positions through the unrelaxed spasm in the affected group.
5. The non-affected muscles frequently refuse to contract due to 'mental alienation'.

"In an exceedingly severe infection, enough anterior horn cells are destroyed at the outset to cause complete flaccid paralysis of certain muscles or parts, and spasm, if present at all, may be fleeting in these cases. Treatment for this condition is ineffective in preventing paralysis" (Cole, Pohl, and Knapp, pp. 7, 8).

"The spasm might even indicate an inflammatory condition of the muscle, as the process appears with the onset of the disease and continues if untreated till the muscle is reduced to a fibrous inextensible mass" (Cole, Pohl, and Knapp, pp. 9,10).

"Spasm is the most damaging finding affecting the muscles in acute anterior poliomyelitis. Paralysis may or may not be present, depending on involvement of the motor cells of the spinal cord, but spasm is the damaging element in the involved muscles."[7]

These statements are given in the repetitious words of the authors in order that no accusation may be made of misrepresentation.

The above statements are, of course, in accord with standard teaching and are not a discovery of Miss Kenny, although no mention is made in the articles quoted that would lead the reader to believe that it was other than Miss Kenny's concept. However, their description of the proved pathology is not sufficiently complete.

Recently, Sabin combined the older knowledge of the pathology and pathogenesis with new discoveries made since 1929. The following is a brief summary of the substance of his article. The earliest observers of this disease (Cornil, 1863, Provost 1865) were able to prove that the major pathological change was in the central nervous system, and no scientific investigator has as yet found any evidence to the contrary. During and since 1929, satisfactory evidence has been adduced that the primary attack in the nervous system is on susceptible neurons, and the inflammatory reaction is the result rather than the cause of the neuronal damage. In the spinal cord, the lesions are more severe and extensive in the anterior horns, but the posterior horns are not altogether spared, and the dorsal-root ganglia almost invariably show destruction of varying numbers of sensory neurons. The motor cortex of the brain may be affected with some regularity. Of particular importance to a proper reinvestigation of the pathological physiology of the disturbance of muscle function in poliomyelitis are the lesions almost regularly present in the vestibular nuclei of the medulla, the roof nuclei and vermis of the cerebellum, and the motor cortex. It may be worth considering that, as regards certain muscle groups, the upper motor neurons concerned in initiating voluntary movement may be significantly and predominantly affected. There is no evidence of any pathological change in the muscles or peripheral nerve during the acute stage of the disease, and any change appearing later is of the type that follows destruction of the motor neurons. The absence of virus from the superior cervical sympathetic ganglia and from the coeliac plexus indicates that these structures do not serve as

pathways of the virus from the alimentary canal to the central nervous system. The irreversible complete destruction of the neuron, and the partial reversible damage to the neuron (in the cases of abortive and transient paralyses) are the pathological unit of poliomyelitis.

This knowledge of the pathological changes in the central nervous system, and an understanding of the symptoms and physical findings which must inevitably accompany them, constitute the orthodox conception of infantile paralysis.

The extent and degree and the transience or permanence of muscle paralysis is dependent primarily upon the damage to the neurons.

Spasm is a reflex manifestation of the peripheral or neuromuscular arc. Irritation of the spinal centers produces hypertonicity of the muscles. This irritation may be due to the inflammation of the spinal cord, or it may be due to an exaggeration of the afferent sensory impulses which excite these centers. It may be due, also, to lesions of the upper, or central, neurons, which normally inhibit the activity of the spinal centers. Any one or all of these factors may be present in the acute stage of infantile paralysis. Spasm, as we all know, is present in many diseases which affect the central nervous system either directly or indirectly. Very definitely, it is not, as stated in the Kenny concept, due to inflammation of the muscles.

Spasm can affect only those muscles for which the spinal centers are in whole or in part intact,—that is, muscles which are normal or partially paralyzed. A completely paralyzed muscle cannot exhibit spasm.

The spasm which is present in the acute stage of infantile paralysis is limited by the factors which produce it. As the inflammation in the spinal cord subsides, the spasm disappears. (It is conceivable, however, that destruction of the central inhibitory factors might prolong spasm indefinitely.) The disappearance of the spasm, with its concomitant pain and tenderness, is generally accepted as evidence that the disease has passed its acute stage and is entering upon the stage of recovery. Evidence of the acute inflammation may last but a few days or be prolonged for three or four months. As the paralyzed muscles regain power, the spasm disappears, which is another way of saying that the stage of recovery has begun.

That there is muscle spasm during the acute stage of infantile paralysis has long been recognized. Recall the work of Wickman and his classification of the forms of the disease as: (1) ordinary paralysis,—anterior poliomyelitis; (2) progressive paralysis,—Landry's paralysis; (3) bulbar paralysis,—polio-encephalitis of the pons; (4) acute encephalitis,—*giving spastic paralysis;* (5) ataxic type; (6) meningitic type; (7) polyneuritic type; and (8) abortive type. It is interesting to note that Wickman recognized the extensive involvement of the central nervous system by this disease, and called attention to the spasm that may be a predominant symptom when the cerebral neurons are involved. The spasm of the posterior muscles of the neck has long been observed as a preparalytic symptom. The author treated many cases of acute infantile paralysis in Philadelphia during the epidemic of 1916, and observed a few cases in which the spasm was so marked and so widespread that a flaccid paralysis could be recognized only after some time had passed.

Miss Kenny's originality, therefore, does not rest upon her discovery of spasm, but upon her interpretation of its cause and its nature, and upon the important rôle that she assigns to it. She apparently fails to recognize its reflex character, and ascribes it to nonexistent inflammation of muscle. Pohl[7] recognizes the temporary presence of this symptom when he says, "during the period of active spasm, usually lasting a week . . ."; but treatment of spasm by hot packs is continued for many months and even for years. The reader is led to believe that spasm is cured by the hot packs, instead of by the natural subsidence of the inflammation of the central nervous system. If it persists for many months in spite of the Kenny treatment, we are at a loss to understand how the treatment usually cures the active spasm in five days and fails to cure it in other cases, or even in the usual

cases, after its application for many months. A rational mind cannot but suspect that the use of hot packs may not have any important effect upon the spasm. When Pohl also says, "Tendency of the muscle to remain in a state of contraction or shortening may persist for weeks or months, and especially if treatment is instituted some days or weeks after the onset", it is quite apparent that he is speaking of the well-known tendency of an unparalyzed muscle to contract and become shortened, when the opponent is partially or completely paralyzed.

"Spasm continues if untreated until the muscle is reduced to a fibrous inextensible mass." [3] But it is well known that, in various types of spastic paralysis, spasm exists for many years without reducing the muscles to a fibrous inextensible mass. To reconcile these phenomena the Kenny proponents apparently are forced to postulate a destructive inflammation of the muscles which are affected in infantile paralysis. "The Kenny method of treatment is based on the belief that poliomyelitis in the acute stage is a disease which attacks not only the nervous sytem but the muscle tissue directly as well. This is an inflammatory process within the muscle, of which spasm is the most distinguishing characteristic." [7] As mentioned by Sabin, this postulate of inflammation of muscle is without foundation. On the other hand, it has been noted by many observers that the fibers of muscles which are paralyzed by poliomyelitis do atrophy and disappear, and that only the fibrous connective tissue of the muscles remains.

Miss Kenny and her followers believe that muscle spasm is the "most damaging finding" for these reasons: (1) The spasm destroys the muscle substance; (2) it produces the deformities and the rigidity of joints observed as sequelae to this disease; (3) it causes "mental alienation" of muscle, a pseudoparalysis occurring in the opposing muscles to those in spasm, in which these opposing muscles are divorced from the voluntary motor pattern and cease functioning; (4) and spasm plus mental alienation causes incoordination of muscle action, resulting in further damage to the motor mechanism.[7]

That muscle spasm causes limitation of motion and deformities of joints is indisputable. This is well illustrated in cerebral spastic paralysis. On the other hand, structural muscle shortening with consequent permanent deformity may be produced by a muscle imbalance caused by paralysis and in the absence of spasm. For example, the peroneal nerve may be injured and paralyzed by a blow or by an incised wound on the outer side of the leg below the knee. The outcome of such a paralysis is exactly the same type of deformity of the foot as is often seen in the final stage of infantile paralysis. It is due to muscle imbalance and not to muscle spasm. To maintain that the deformities of infantile paralysis are due largely or entirely to muscle spasm is to ignore the fact that a real injury of the anterior-horn cells occurs at all, or that it is a common occurrence. Pohl states, "paralysis is unfortunately a feature of the disease, but paralysis proves after all to be a minor consideration in most cases of infantile paralysis". Contrast this statement with the proved facts: "The irreversible complete destruction [of the neuron] as well as . . . the partial reversible damage to the neuron [are] the *pathologic unit* of poliomyelitis".[9]

Schwartz and Bouman state, "The spasticity present in acute infantile paralysis is of a reflex nature and is not present in the completely paralyzed muscle. It exists in the weakened muscle and in its antagonist and in muscles in parts of the body in which clinical symptoms of disease are not evident. . . . When the strength of voluntary contraction increases through treatment, the spasticity decreases." Therefore, only normal muscles or partially paralyzed muscles can exhibit spasm. Completely paralyzed muscles must be flaccid. This is in accordance with the orthodox teaching that the destruction of the anterior-horn cells produces a flaccid paralysis. Miss Kenny states, "The flaccid muscles are normal. Loss of ability to contract these is due to functional dissociation (alienation) from the nervous system" (Pohl and Kenny, p. 37). It is upon the basis of this false concept that she maintains that orthodoxy has largely been devoted to treating normal muscles, but it is admitted that, "in an exceedingly severe infection enough anterior-horn cells

are destroyed to cause complete flaccid paralysis of certain muscles".[3] How, therefore, by the Kenny concept is one to distinguish between the flaccidity which is due to the motor-cell destruction and that which is due to "functional dissociation (mental alienation)", when the only characteristics of these muscles are flaccidity and paralysis?

If all muscles are in spasm, the condition would resemble that found in cerebral spastic paralysis. Deformities would occur because certain muscles which are primitively stronger (adductors, flexors) overcome their weaker opponents, as if they were at a "tug-of-war". But these deformities develop slowly and do not become permanent for a long time. In infantile paralysis, a completely paralyzed muscle, which can exhibit no spasm, must very quickly be overcome by a normal muscle which is in spasm, and a weakened (partially paralyzed) muscle, even though in spasm, must be readily defeated by its normal (unparalyzed) opponent. The rapid development of a deformity, such as a foot-drop, in infantile paralysis can be explained only on the basis of a complete or partial paralysis of the anterior muscles of the leg. According to the Kenny concept, the "affected" posterior muscles overcome and overstretch the normal anterior ones; but, in all reason, it is the strong which overcomes the weak. The anterior muscles of the leg, if normal, overcome weakened posterior muscles and produce a talipes calcaneus. If Schwartz's observations are correct, the "affected" muscles are the normal ones and the "normal flaccid (but alienated)" muscles are the paralyzed ones. However, Miss Kenny states (Pohl and Kenny, p. 22), "Dr. Schwartz of Rochester, New York, has made scientific research with regard to the condition of muscle spasm, and the results have proved my concept correct". She seems not to understand the logical conclusions of Dr. Schwartz's observations.

Let us now examine her method of treatment of infantile paralysis, which is stated to consist of the avoidance of splinting of any kind, the use of hot packs in a very particular way, and a system of muscle re-education. We are warned that every detail of the treatment must be rigidly followed, in order that the best results may be obtained.

Consider briefly the principles and the methods of the standard or orthodox treatment, in order that the Kenny methods may be contrasted with them. By orthodox methods are meant those methods which have been taught in medical schools and which are found in standard textbooks. This does not imply that these methods have been universally and rigidly followed. There has undoubtedly been ignorance or carelessness of some medical practitioners; there are, of necessity, some circumstances, for example, a lack of trained nurses and physiotherapists, which prevent the most efficient application of these principles and methods. On the other hand, the principles of treatment are simple and explicit, and the methods of treatment can be efficiently used by the parents of the child. No elaborate, time-consuming, and expensive regimen is necessary.

Rest is the most important principle of treatment during the stage of acute inflammation. The value of rest in the presence of inflammation was emphatically taught by John Hilton eighty years ago, and has been an accepted principle of medical practice ever since. The central nervous system can be put at rest physiologically rather than anatomically by preventing or diminishing the function of the sensory and the motor apparatus. When activity of the motor cells is diminished, they are placed at rest. This may be accomplished by immobilization of the skeletal muscles. Inasmuch as the motor centers of the spinal cord are reflexly stimulated by sensory impulses from the periphery, these exciting sensory impulses should be abolished as far as possible. Spasm is due to over-excitation of the reflex arc which consists of motor cells and their efferent nerves, the muscles and their tendons, and the afferent sensory nerves; and stimulation of any one of these three elements increases the activity of the entire arc. Sensory impulses are diminished by controlling pain, by handling tender muscles gently, and by moving the extremities as little as possible. It has long been known and taught by the medical profession that the application of heat exercises a soothing influence and tends to relieve pain, and consequently to allay excitation of the peripheral, or reflex, arc. Immobilization and the use of heat are,

therefore, the logical principles of the treatment of infantile paralysis during the acute stage. Furthermore, it is recognized that the immobilization should not only place the neuromuscular apparatus at rest, but it should also prevent the early appearance of deformity which arises from muscle imbalance, the effect of gravity, the weight of bedclothes, and faulty positions of the recumbent patient. Well-molded posterior plaster splints, or others, such as the "Toronto splints", well-padded, making no pressure, and, entirely comfortable, have generally been employed. Prolonged fixation in complete plaster casts has not been recommended by standard authorities, for very evident and well-known reasons.

The following quotation from the standard textbook on Orthopaedic Surgery by Jones and Lovett (p. 447) confirms the statement that this practice is orthodox teaching. "Treatment should consist of rest and absence of irritation, and the avoidance of meddlesome therapeutics. Fixation of the neck, trunk and limbs in some comfortable appliance such as a well padded Plaster of Paris bed is desirable in the most acute cases. . . . Immersion in a warm saline bath is agreeable and apparently beneficial toward the end of the tender stage and may be comfortably carried out by immersing the patient on a sheet. It is not physiological to irritate and stimulate the peripheral ends of nerves connected with affected and hemorrhagic nerve centers by massage and muscular exercise while the acute process, as evidenced by tenderness, exists. Joints will not become ankylosed, muscles will not hopelessly atrophy, and the patient will not become bedridden because he is kept quiet for as long a time as need be, to enable the damaged cord to repair without interference."

The Kenny method emphasizes, as does the orthodox method, the importance of handling the patient gently. "Care must be exercised not to aggravate the condition of spasm by frequent examination or rough handling of the patient. Extreme gentleness in nursing attention is required in order not to cause more pain and spasm in the irritable muscles."[7] This quotation contains an element of truth, but it illustrates the constant effort to exalt "spasm" as the essential and the outstanding feature of the disease. Rough handling may increase spasm, but it produces this reaction because it excites irritable motor cells in the spinal cord, whose function is to produce muscle contraction. The disease consists of the inflammation of the central nervous system and not of the spasm of muscles.

Under the Kenny method, the patient is put to bed on a firm mattress. A footboard keeps the feet dorsiflexed to a right angle and removes the weight of the bedclothes from them. The knees and the hips are maintained in extension and the patellae point to the ceiling. "The patient is placed in a position as closely approximating the normal standing position as possible. . . . If spasm is severe, modifications of this position may be necessary to allow relaxation of the spastic muscles. . . . The purpose of the footboard is to allow the patient to maintain the normal standing reflexes during his stay in bed and it is not in any sense a splint" (Cole, Pohl, and Knapp, pp. 18, 19). "The Kenny method particularly stresses the avoidance of any form of immobilization." It "employs no splinting of any kind. On the basis that spasm is the state affecting the muscles, it follows that splints would tend to aggravate the irritable muscles and increase the spasm. Rigid immobilization also tends to encourage shortening of muscle and resulting permanent contractures, *et cetera*".[7] These statements are contradictory and illogical. They condemn immobilization and yet maintain the patient in a meticulously described position on a firm bed. A device that holds the foot at a right angle to the leg is declared vigorously not to be in any sense a splint. A splint is a device to maintain parts of the body in a given position. If the child moves his foot away from the footboard, with or without permission, the board is, of course, in no sense a splint; but, in such case, why should they use it to maintain a prescribed position of the foot? If it does not act as a splint, it serves no useful purpose. It is maintained that the board stimulates the normal standing and postural

reflexes. Are these reflexes not those which preserve the balance of the body in stance and in locomotion? How they can be stimulated when a patient is flat on his back is difficult to understand. Why should a footboard stimulate (?) these reflexes more than a splint that presses against the sole of the foot? Why should a molded, well-fitting, softly-padded splint that distributes pressure on the body over a larger area "aggravate the irritable muscles and increase the spasm", when a footboard and a firm mattress do not do so? It can be stated positively that rest allays irritation and likewise spasm, insofar as spasm is dependent upon peripheral irritation. A proper splinting is the most effective means of putting the parts at rest. The Kenny method of immobilization is less effective and it does not completely fulfill its announced purposes.

Miss Kenny accuses orthodoxy of permitting the "flaccid normal" muscles to become paralyzed through overstretching, and yet denounces the use of splints which orthodoxy has employed successfully to prevent this disaster. Standard teaching has always emphasized the necessity of the prevention of overstretching of muscles, particularly of the weak and paralyzed ones. "If there is active spasm in the posterior calf muscles, the foot is not put in contact with the footboard until the spasm has been released" (Cole, Pohl, and Knapp, p. 19). In this case the anterior muscles, whether they be normal, weakened, or completely paralyzed, are permitted to be overstretched, because to maintain the foot at a right angle to the leg would aggravate the spasm. It is well known that a sudden stretching of a spastic muscle increases the spasm, as in the elicitation of ankle clonus. Schwartz and Bouman, in the article already quoted, observed this phenomenon in acute infantile paralysis. It is equally well known that gradual continuous stretching will overcome spasm and secure elongation of the muscle, and that the elongation can be maintained by fixation of the parts. The foot can be maintained at a right angle or beyond, without increasing spasm or permitting it to overstretch weak opposing muscles and produce deformity. It is curious how "spasm" is encountered at every turn of the Kenny concept and method. While only a symptom, it has been exalted to the dignity of a newly discovered disease; it destroys muscles which it affects and the opposing unaffected muscles; it causes all the crippling deformities; it runs its vicious course through the weeks and months and years unless or until it is subdued and eradicated by the Kenny hot packs, the only weapon which is effective against it. These ideas might well be called an obsession.

The treatment of acute infantile paralysis by the use of hot packs is the main feature of the Kenny method. All details of their application are meticulously described; the material from which they should be made (which is what Miss Kenny happened to have on hand when she began her work in Australia), their size and shape, the frequency and duration of their application, what parts of the body should be covered and what parts left exposed, and how the bedding must be protected from the moisture. Incidentally a device has recently been advertised by a leading manufacturing company, a newly invented clothes wringer especially adapted to wringing Kenny hot packs. We are admonished that unless the Kenny technique is accurately followed, the treatment may not be successful.

That a judicious application of heat promotes circulation and the nourishment of the soft structures of the body, that it may exert a soothing and relaxing effect and relieve pain, has long been known. Heat is used during the acute stage of infantile paralysis for precisely these reasons, when it can be applied in a manner which does not increase the irritation of the central nervous system. It might be pointed out that a light, gentle, rhythmical stroking massage in many conditions may produce the same quieting and soothing effect as heat. While moist heat is preferable to radiant heat in some conditions, it does not appear reasonable that hot packs work a miracle in the treatment of acute poliomyelitis when immersion in warm water or the use of radiant heat completely fails. That heat applied in any form or by any method to the trunk and the extremities can affect the action of the virus of poliomyelitis upon the central nervous system is not credible

in the light of our present knowledge of this disease. The Kenny concept of the value of hot packs is dependent upon the false assumption that there is present an inflammation in the muscles themselves. If this premise were correct, there might be some validity to her conclusion.

Let us now consider the treatment of infantile paralysis during the stage of convalescence, or recovery. This stage begins when muscle tenderness has disappeared and joint movements may be made without pain. Passive and active movements are begun. Weak and paralyzed muscles are protected from prolonged overstretching and from overwork. Careful muscle re-education is begun immediately and is continued for as long as muscles continue to improve. Contractures and deformities are prevented as far as possible by necessary splinting while the patient is recumbent in bed, and by proper braces or appliances when he becomes ambulatory. The extent and the degree of recovery are, in the last analysis, dependent upon the recovery of the anterior-horn cells. As already quoted from Sabin, the damage to the neurons is in some instances irreversible, or permanent; in other instances it is reversible. In the latter, the recovery may be so complete as to eradicate spontaneously all evidence of paralysis, or it may be incomplete and be manifested in a partial permanent paralysis or weakness of certain muscles. In such a case, some of the neurons supplying a muscle have recovered; others have not. No known treatment affects the reversibility of the damage to the neurons after they have been attacked by the virus of this disease.

Despite the truth of these last statements, there are rational grounds for treatment during the stage of recovery. Overstretched and elongated muscles cannot respond adequately to the increasing energy discharged from the motor cells as they recover their function. Nor can they contract when their movement is prevented by a structural shortening of their opponents. Hence the necessity to prevent overstretching and contractures. Muscles which are completely or partially paralyzed at the end of the acute stage of infantile paralysis are atrophied, and contract feebly to any method of stimulation. Muscle re-education aids in increasing the size and strength of the muscle fibers during the period of recovery, and in regaining cerebral control of muscle movements. Any muscle, normal or enfeebled by disease, can be made stronger by exercise. Likewise can the cerebral control of muscles be perfected.

Observe now the striking similarities of the orthodox and of the Kenny methods of muscle re-education, and the utter diversity of the reasons given to justify the methods. The quotations marked (a) are taken from Jones and Lovett's textbook on Orthopaedic Surgery and those marked (b) are from the pamphlet of Cole, Pohl, and Knapp.

(a) "Muscle training is fundamentally an attempt to restore a cerebral motor impulse to a muscle, an impulse which has either been impaired or lost during the acute stage of poliomyelitis. The method of muscle training aims at two things: first, at forcing the efferent impulse to develop a new path around the disordered nerve center in the cord; and second, at securing contraction of the desired muscle, however feeble, which is of course the best possible treatment of the muscle itself. These two objects rest upon a sound anatomical and physiological basis." (b) "In addition to anterior horn cell destruction the lack of function is considered also to be the result of loss of connection with the central nervous system either through 'mental alienation' by which the impulse is suppressed or through 'incoordination' by which it is diverted to other channels, or both. The purpose of re-education is to restore connection of the part with the central nervous system (to restore 'mental awareness'). Muscle strength is not a primary consideration but the re-establishment of 'awareness' and the production of a normal rhythmic motion no matter how weak is the aim of treatment. Increase in muscle strength will follow." Both speak of the loss of the connection of the muscle with the central nervous system. Jones and Lovett base this upon the known pathology of the disease. Miss Kenny attributes it to an obscure psychological process which she terms "mental alienation" or, in the next

breath, to a physiological process of "incoordination" by which an impulse is diverted to other channels, or both. She explains and elucidates neither the psychological suppression nor the physiological diversion of cerebral impulses. It is to be presumed that by "incoordination" she means "muscle substitution" and "mass movement"; but we are returned definitely to the field of psychology when she declares that the restoration of "mental awareness" is the purpose of muscle re-education.

(a) "If a muscle is apparently without any power, the patient should concentrate his attention on the attempt to accomplish the movement while it is performed passively." (b) "The Kenny method of muscle re-education is based on the symptoms and concept of the disease previously described. First, it is assumed that nerve cells are not permanently or completely destroyed until failure of the muscles supplied by these cells to respond to treatment indicates that destruction has occurred." Both are in agreement that it is impossible to determine at the beginning of the stage of recovery whether a paralyzed muscle will recover or not recover, and that treatment should be given on the assumption that it can recover. In the Kenny concept, however, paralysis is said to be a minor feature of the disease, but her treatment by muscle re-education is directed toward the cure of the paralysis. It would seem that the muscles which formerly were called normal but "alienated" are now treated as if they were paralyzed because of damage to the anterior-horn cells.

Both methods insist that:

1. The patient concentrate his attention upon his efforts to make a prescribed movement;
2. The movement be rhythmical;
3. The joint be moved through its entire range of motion;
4. Fatigue of a weak muscle be avoided;
5. The muscle re-education be most carefully supervised and directed by a competent person;
6. Muscle substitution or mass movement or incoordinated action be prevented;
7. The application of heat before exercises are given is useful.

(a) "The patient should concentrate his attention on the attempt. . . . It often happens that weak muscles may be able to carry the limb through only part of its normal arc of motion. In such a case the limb should be carried . . . through the remaining arc of motion normal to the joint, and there should be no pause after the muscle has ceased acting, but the assistance should come in time to make one smooth movement throughout the whole arc, in order that there may be no interruption in the patient's mental effort." (b) "The patient has passively full range of motion in all his joints, . . . and most of them are more limber than they were before the onset of the disease." (One might inquire how it is known that they are more limber than before the onset of the disease.) "This harmonious action which allows a smooth, orderly and effective functioning of muscles is well referred to as coordination. . . . A muscle, whether paralyzed or alienated, which is being trained must be made to contract, in a mental sense if in no other way; the conscious mind must accept this 'awareness' of the muscle if orderly and coordinated action is to be obtained." (a) "Not until muscles are capable of performing a movement through the whole arc should any additional resistance be given, and not until they are capable of performing the movement next in order of strength throughout the whole arc, should that movement be given in place of the easier one. . . . Each exercise [should be given] in slow enough rhythm to allow for complete recovery between efforts. Unless it is done as well the second time as the first, and the tenth as well as the second, it is being done too fast or continued too long and the patient should be given a rest at once." (b) "Care is taken also not to tire the patient in any way. Treatment must be discontinued if the patient shows signs of fatigue or is uncooperative for any reason. As the patient learns

how to move the muscle more movements are given, gradually increasing the range and number of motions, although enough to tire the muscle is never allowed." (a) "If possible the patient should never be left to do his exercises alone, even when he is old enough to understand his own case. The response of nerve and muscle is dependent upon the stimulus, and the volition of the patient is greatly aided by the outside stimulus of a word of command. . . . Accuracy and precision are of the utmost importance in obtaining a proper result. Carelessly performed exercises are of little value and loosely prescribed exercises harmful because if some of the muscles are weaker than others, unless the exercises are carefully limited, the tendency of the patient is to use the stronger rather than the weaker muscles and thus to increase the disparity between the two. . . . In all exercise periods the whole attention of the patient should be required." (b) "In the presence of incorrect muscle action such as substitution or incoordination active movements on the part of the patient are prohibited until passive motions can be carried out by the technician with complete relaxation of the patient. Having achieved this state, voluntary motion on the part of the patient may be instituted but such action is always under the control and guidance of the technician."

The use of heat during the convalescent stage of infantile paralysis has long been advised and employed. "If a partly paralyzed limb is heated it is capable of performing better muscular function than before, because muscles work better at a higher temperature. . . . [It] adds to the effectiveness of massage. . . . Heat may be applied as radiant heat or non-radiant heat. Hot water is not desirable as a form of heat, as it makes the skin tender and cannot be borne at so high a temperature as can dry heat" (Jones and Lovett, pp. 453, 454). Exercises in warm water to remove the resistance of gravity and make it possible for feeble muscles to move the limb have been extensively used. The Kenny hot packs, presumably, have an effect, similar to the application of heat by other methods. It is inconceivable that one method should secure muscle curative effects while the other fails dismally.

It is evident, therefore, that the Kenny methods of treatment are very similar to the orthodox methods, and in many particulars identical with them in spite of the different terminology employed by Miss Kenny. The statement [8] that the two methods have nothing in common displays an ignorance of the orthodox methods. The one distinctive feature of the Kenny method of treating infantile paralysis is the use of the hot pack to relieve spasm. That it does eliminate the reflex spasm of the acute disease has not yet been proved. That it can do so is not logical except insofar as it may exert a favorable effect upon the pain and tenderness, and thus diminish the afferent impulses that excite reflex neuromuscular activity. That it can destroy the virus of the disease or affect in any way its destructive action upon the central nervous system is not credible.

The end results secured by one method of treatment or another are the final criteria of its value. Miss Kenny states that deformities are practically abolished and a much higher percentage of recoveries is obtained than by any previous method (Pohl and Kenny, pp. 25, 37). She has stated repeatedly in public that 12 per cent. of patients afflicted by this disease recover under orthodox treatment and 80 per cent. or more under the Kenny method. The figure of 12 per cent. (Pohl and Kenny, p. 24) is taken from the statistics of McCarroll and Crego which do not include the large number of cases which have the abortive (non-paralytic) type of the disease or many of the equally large number which make a spontaneous recovery from paralysis as the reversible damage to the neurons is cured by natural processes. The 80 per cent. cured by the Kenny method (a claim not supported thus far by the publication of any reliable statistics) presumably includes these two large groups.

A personal communication received from the Department of Health of the Commonwealth of Pennsylvania states that in Pennsylvania in 1941 there were 741 cases of acute poliomyelitis. Of these fifty-six patients died and 54 per cent. of the remainder recovered

without paralysis, 19 per cent. being abortive cases and 35 per cent. making a spontaneous recovery.

Lovett stated, "we should bear in mind that practically all patients improve after the acute attack is ended, that about 25 per cent. recover spontaneously . . . and that no one can foretell in a given case how rapid or how slow the improvement will be. Under these circumstances loose generalizations as to any form of treatment are likely to be misleading, and only those based on a very extensive clinical experience are likely to be of value." Furthermore, the differences in the effects of the disease in different individuals, in different communities, and in different epidemics make an accurate analysis of end-result studies exceedingly difficult or even impossible.

The claim that the patients treated by the Kenny method recover without residual deformities is unsubstantiated and has been denied by numerous observers.[2] Muscle imbalance is certain to result in deformities even though they may not arise until the patient has become ambulatory. There must be muscle imbalance when there is a residual paralysis.

The propaganda for the Kenny concepts and methods of treatment has been harmful in that it has sought to cast discredit upon scientific medicine and the medical profession, in that it holds out false hopes to the victims of infantile paralysis and their families, and in that the method of treatment is expensive and is consuming of time and labor. The money could be better spent in increasing our scientific knowledge of this disease. The labor and the time could be better employed in the physical rehabilitation and in the education and the occupational training of the crippled.

The author believes that the Kenny furor will eventually subside, and that it will go the way of the countless fads for the cure of disease that have preceded it, because only that which is built upon the sound foundation of truth can stand the test of time.

REFERENCES

1. Annual Report of The National Foundation for Infantile Paralysis, 1942.
2. Blount, W. P.: Much Ado about Polio. Milwaukee Med. Times, XVI, 15, 1943.
3. Cole, W. H.; Pohl, J. F.; and Knapp, M. E.: The Kenny Method of Treatment for Infantile Paralysis. New York, The National Foundation for Infantile Paralysis, Publication No. 40.
4. Jones, Robert, and Lovett, R. W.: Orthopaedic Surgery. New York, William Wood and Co., 1923.
5. McCarroll, H. R.: The Role of Physical Therapy in the Early Treatment of Poliomyelitis. J. Am. Med. Assn., CXX, 517, 1942.
6. McCarroll, H. R., and Crego, C. H., Jr.: An Evaluation of Physiotherapy in the Early Treatment of Anterior Poliomyelitis. J. Bone and Joint Surg., XXIII, 851, Oct. 1941.
7. Pohl, J. F.: The Kenny Treatment of Anterior Poliomyelitis (Infantile Paralysis). Report of the First Cases Treated in America. J. Am. Med. Assn., CXVIII, 1428, 1942.
8. Pohl, J. F., and Kenny, Elizabeth: The Kenny Concept of Infantile Paralysis and Its Treatment. St. Paul, Bruce Publishing Co., 1943.
9. Sabin, A. B.: Pathology and Pathogenesis of Human Poliomyelitis. J. Am. Med. Assn., CXX, 506, 1942.
10. Schwartz, R. P., and Bouman, H. D.: Muscle Spasm in the Acute Stage of Infantile Paralysis. As Indicated by Recorded Action Current Potentials. J. Am. Med. Assn., CXIX, 923, 1942.
11. Wickman, Ivar: Die akute Poliomyelitis bzw. Heine-Medinsche Krankheit. Berlin, Julius Springer, 1911.

EXPERIENCES WITH THE KENNY TREATMENT FOR ACUTE POLIOMYELITIS IN THE EPIDEMIC OF 1942, MONMOUTH AND OCEAN COUNTIES, NEW JERSEY *

BY NICHOLAS S. RANSOHOFF, M.D., NEW YORK, N. Y.

"To be or not to be" appears to be the question in the mind of every medical man who is eyeing the Kenny method of treating poliomyelitis, with his reactions veering from the venomous to the ecstatic. Is it to be Kenny, or not? As yet there seems to be no happy medium. Either it is regarded as a crusade, or else it is roundly condemned as rubbish. Neither attitude, in the estimation of the author, appears to be the right one. The treatment is still being given a thorough trial, which has not been completed. Therefore this is a *preliminary* report.

This report is based on actual experiences at the Monmouth Memorial Hospital, Long Branch, New Jersey, and on the treatment of patients of Monmouth and Ocean Counties who had infantile paralysis in the late summer and fall of 1942. A general summary of what has been accomplished in these cases will be compared with what was accomplished in the previous ten years at this same institution, with patients from the same regions, by the same team of orthopaedic surgeons and physiotherapists. In these years, the so-called orthodox method was employed. It is realized that this term covers a multitude of sins, but the figures show that these patients certainly were not sinned against.

All of the patients admitted to the Hospital in the fall of 1942 were treated with the so-called "Kenny method". It is advisable to give a picture of the exact conditions under which these patients were treated.

In the spring of 1942, the head of the Physiotherapy Department of Monmouth Memorial Hospital was sent to Minneapolis to take a course with Sister Kenny. She took the full course, plus an additional month. She came back full of enthusiasm for both Sister Kenny and her method, and was a most ardent advocate and crusader for Sister Kenny. Dr. Lehmacher, the head of the Physiotherapy Department at Paul Kimball Hospital, Lakewood, was also sent to Minneapolis and took the course for physicians. In anticipation of the usual number of patients with acute anterior poliomyelitis, equipment was set up, and student nurses were trained to handle about eight patients. Thirty-one patients were admitted, and, with excellent cooperation on the part of the hospital administration, it was possible to care for all of these patients by means of the "Kenny method" as then understood. It should be stated at the outset that there were handicaps, caused by lack of personnel and experience; therefore, certain details were not carried out meticulously. It is certain that these deficiencies have not been sufficient to change materially the progress of any of these patients. There were times when some of these patients did not get sufficient packs, and there were others when patients did not receive adequate muscle training. However, these are the patients who have done very well indeed and have been discharged without paralyses. Patients who were very sick *did* receive adequate packs and adequate muscle training, but they were not treated "rough" enough,—that is, too great care was taken in handling them.

In the previous ten years, twenty-four cases of poliomyelitis were admitted to the Hospital. Five patients were put in respirators; of these, four died and one lived for about three years with severe residual paralysis. Of the remaining nineteen patients, five made a complete recovery of all their paralyzed muscles; there are four others who are still under treatment and in whom the end result cannot yet be determined. These four still have some muscle weakness. Two patients were lost track of after they left the Hospital, so that the end results are not known. There are eight in whom some permanent residual paralysis remains.

* Read at the Annual Meeting of The American Orthopaedic Association, Cleveland, Ohio, June 7, 1943.

Before proceeding farther with this discussion, it is necessary that the term "cure" be defined. In all of the cases which are discussed, "cure" will be used only in reference to those patients who have recovered from their acute anterior poliomyelitis with no paralysis in any muscle. That is the only definition of "cure" which is justifiable. It has been impossible, both from talking to Sister Kenny and from reading her books and articles, to get a clear conception of what she or her advocates mean by the term "cure". Sister Kenny told the author that there has been no case of acute poliomyelitis, that she has treated, where it has been necessary to put the patient in a crippled children's home, or where the patient has not been able to return to school or to his previous occupation. *That classification is not good enough.* There have been few patients that any one here has treated, who could not be considered cured under this definition. Certainly there have been a great many things that this writer has learned from Sister Kenny. Her concept of the part that spasm plays in this disease is most certainly a new one; the existence of spasm over a long period of time and its deleterious effects were never understood nor appreciated. The emphasis on incoordination, mental "awareness", and "alienation" is also an important factor in a better understanding of the treatment of these patients. Sister Kenny demonstrated a patient who the author thought had a pelvic obliquity and weak abdominal muscles. In less than two minutes, Sister Kenny showed that this apparent paralysis was due to the spasm of the quadratus lumborum; she further demonstrated the inability of the abdominal muscles to work when stretched. She then "released" the spasm in the quadratus lumborum, bringing the abdominal muscles back to their normal resting length, and overcame the incoordination. The patient thereafter was able to use the abdominal muscles, and to hold himself in typical "V" position. This certainly could not have been accomplished with weak abdominal muscles. She also demonstrated satisfactorily the fact that incoordinated hamstrings could not flex the knee, and that, by restoring the "mental awareness" and coordination, the patient was able to flex the knee without difficulty.

There would seem to be very little doubt that the hot packs give the patients a good deal of comfort, but it should not be stated as yet that they have further therapeutic value.

The spasm or tightness most certainly lasts a long time, even months. This tightness or spasm is worse when the patients are handled too gently. One of the errors was in handling the children too much like fragile dolls, rather than making them move themselves, and in neglecting to stretch the muscles that tended to tighten. This lesson has been learned and the error will not be repeated. The muscles are easier to stretch after the hot packs; whether this means that spasm has been released is difficult to say.

In discussing the experiences which were had in the epidemic of the summer and fall of 1942, it should be emphasized that this is purely a preliminary report. It is realized that no final report can be made on any epidemic until at least three years have elapsed. The following are some of the observations resulting from our work during the past epidemic.

A number of patients were seen, who, under ordinary circumstances, would never have been admitted to the hospital, who would not have been seen by an orthopaedic surgeon, and whose condition would not have been diagnosed as poliomyelitis, had it not been for the fact that the entire community was "polio-conscious". This apprehension was engendered by the newspaper releases of The National Foundation for Infantile Paralysis. Spasm was found in every one of these cases, but there were seven who never had any real paralysis. Diagnosis was made on the spasm of the neck and back and hamstring muscles, on muscle tenderness, and on high cell counts in the spinal fluid.

The cases that were seen in this epidemic have grouped themselves into several distinct patterns. These patterns include:

1. The patients with non-paralyzing poliomyelitis, as just described.

2. Patients having poliomyelitis with generalized severe muscle tenderness and muscle spasm, in whom the muscle spasm, tenderness, and pain were relieved over a long period of time. These comprised five, of whom two still have spasm, and still have muscle tenderness; in the early stage they showed no real muscle weakness, but have gradually gone on to deterioration of muscle strength.

3. Patients with frank paralyses of varying extent, from isolated muscle groups to entire members.

4. Patients with poliomyelitis of a severe bulbar type, of whom there were five. Four of these patients died, three within thirty-six hours of their admission. In all of these cases, Kenny packs were tried but were unavailing. The patients were then put in respirators; in three of them the respirator had no effect, but in two it was apparently of value. One of these seemed to be doing very well indeed yet died suddenly at the end of six weeks, after he was virtually out of the respirator for two weeks; he developed respiratory difficulties, and, in spite of being put back into the respirator, died. The fifth patient is doing well; he has been out of the respirator for four months; there are residual paralyses and contractions, but the boy is improving and his treatment is being continued.

In the present series, thirty-three cases are reported. Of these, four have died, and one has been lost track of because the parents refused to bring the child in for further treatment. He is now being taken care of by another doctor, with heavy braces and crutches which he does not use. There are seven patients with severe paralyses, eight who have mild residual paralyses, and thirteen who are completely cured. The dividing line between mild and severe is always a difficult one to draw, and there is no question in the mind of the writer that the statistics in these cases will change before the end results can be shown.

What, then, can be reported as our present opinion in reference to these experiences with the Kenny method?

1. It was determined at the outset to try the Kenny method for three years; this is being done.

2. In our community, at least, due to newspaper publicity, parents have demanded the Kenny treatment, and no other.

3. The cost of Kenny treatment, thoroughly carried out, is tremendous. Unfortunately, because of clerical shortage, the hospital administration has not been able to supply accurate comparative figures. A personal estimate would be that the Kenny treatment is at least five times as expensive as the so-called orthodox treatment.

If, however, it can be proved that this method is superior, then the question of cost is not a consideration.

4. It can be asserted that no patient has suffered ill effects because of treatment by the Kenny method. For a time, before the importance of stretching tight muscles was appreciated, there was considerable concern that fixed deformities might develop. After Sister Kenny demonstrated the proper handling of this problem, there have been no further difficulties to date.

(By the way, there will probably be objections made to the term "stretching tight muscles". By this is meant the putting of parts through their complete range of motion, and attempting to simulate physiological movement.)

5. The experience gained in the epidemic of 1942 will make the treatment of patients better in subsequent years.

6. It is felt that the moving of parts through complete ranges of motion has been of decided benefit from a physiological point of view. This increases circulation, movement of lymph, *et cetera*.

7. In a discussion of the Kenny treatment at the Hospital for Joint Diseases, the author said, "Throw away your braces, but not too far". It was interpreted as condemn-

ing the method, by those who heard this remark. It was *not* meant as condemnation, but was merely a statement that all patients will not make complete recoveries, and that braces *will* be needed.

8. Sister Kenny has made a real contribution. She herself, like all originators of a method, reports results which we have not yet been able to duplicate. She has been endowed with a great gift in the healing art. There is still a question as to whether or not she can impart this gift to her disciples. Certainly, through her, a large number of people are being trained in an excellent method of muscle re-education. For this, if for nothing else, all who have to do with the therapy of poliomyelitis must be most grateful.

How much of the Kenny concept will remain intact is not known. Many of her ideas of kinesiology are still difficult to accept. Others are so simple and apparent that it seems incomprehensible that they were not recognized long ago.

In conclusion, therefore, the author reserves his opinion until further experience demonstrates the exact value of the Kenny method. Then only may a final report be given.

ANALYSIS OF NEUROMUSCULAR DISORDERS IN POLIOMYELITIS *†

BY JOSEPH MOLDAVER, M.D., NEW YORK, N. Y.

From the Department of Neurology, Columbia University, College of Physicians and Surgeons, and the Neurological Institute of New York

The neurotropic virus of poliomyelitis has a marked affinity for the great anterior-horn cells of the spinal cord. Lesions are found mostly in the cervical and lumbar enlargements, where many of these cells are located. These injuries explain the usual localizations of the motor deficiency. If the lesion is marked and extensive, it will lead to complete wasting of the corresponding muscles. Slight and transitory damage of anterior-horn cells explains the reversible paralysis and spontaneous recovery which are common in this disease. Destruction of a small number of motor cells may fail to produce clinical effects, and sometimes, therefore, may be overlooked. The study of cases during epidemics, as well as experimental work on monkeys, has shown that lesions are not confined to the lower motor neuron alone, but that many parts of the central nervous system may be affected. Injuries of dorsal-root ganglia, posterior horns, posterior roots, and the sympathetic column are common findings. Therefore, in addition to the motor deficiency, meningeal signs, hyperaesthesia, pain, and tenderness of the muscles exist in almost every patient. Pain is increased by any attempt to stretch the muscles, and may be found in affected muscles as well as in non-paralyzed ones. This symptom might be explained as a referred pain, due to lesions of the sensory protoneurons. Inflammation of the dorsal-root ganglia, the posterior horns, or the posterior roots is not necessarily found at exactly the same level as the lesions of the anterior-horn cells. Therefore, it is not surprising that some paralyzed muscles are not painful, while normal and non-degenerated muscles may show marked pain. The paralysis is a flaccid one; the muscles become atrophic and toneless; the tendon reflexes and some cutaneous reflexes are absent. In the weak or paralyzed muscles, the existence of neuromuscular degeneration can easily be demonstrated; it is the result of lesions of the lower motor neurons.[7] Clinically, most of the lesions of the sensitive protoneuron clear up completely.

The Kenny concept recently described [6] is fundamentally different. The phenomena described in this concept are: (1) muscle "spasm", (2) "mental alienation", and (3) "incoordination". Muscle "spasm" is regarded as the most damaging symptom in poliomyelitis; and is said to lead to degeneration, if not treated. The danger of paralysis lies mainly in allowing "spasm" to continue. Infantile paralysis is a "spastic" and not a flaccid paralysis. "Spasm" is described as being generally present in the following muscles: neck, back, hamstrings, calf, and pectoralis. The muscles opposed to those in "spasm" become "mentally alienated",—"divorced from the patient's mind". "Alienated muscles appear toneless and incapable of voluntary contraction, but are never painful or tender, indicating that they are not the muscles involved directly by the disease" (Pohl and Kenny, p. 42). "Even the tendon reflexes of such muscles disappear; alienated muscles become permanently paralyzed and atrophied, if steps are not taken early to restore such muscles to action" (Pohl and Kenny, p. 52). The dissociation of the muscle from the brain is regarded as due to some physiological block. The third symptom described, "incoordination", is due, according to the concept, to a "disorganization of the regulating centers".

To test this concept, a series of investigations have been carried out, since obviously

* Read in part at the Annual Meeting of The American Orthopaedic Association, Cleveland, Ohio, June 7, 1943.

† This study was sponsored by The National Foundation for Infantile Paralysis, Inc., New York, N. Y.

a clear understanding of the symptoms is of great importance for the diagnosis, the prognosis, and the treatment of any disease.

TECHNIQUE EMPLOYED

Neuromuscular degeneration, such as would be caused by lesions of the anterior-horn cells, was explored by the chronaxia method; and so-called "spasm" was studied in some patients, mostly by electromyograms.

Without entering into details, it might be useful to recall briefly the significance of chronaxia and of action potentials.

Chronaxia (from *chronos* meaning time, and *axia* meaning measure) is a measurement of excitability. It is based upon the fact that the strength of a stimulus applied to initiate a response is not sufficient to characterize excitability. The stimulus must last some time. The chronaxia is defined from the galvanic threshold called rheobase (from *rheos* meaning current, and *base* meaning basis). It is the duration necessary to obtain the threshold, when an intensity of twice the rheobase is used.

The chronaxia of the normal skeletal muscles measured at the motor point with small electrodes varies between .06 and .50 millisecond, depending upon the muscle. For instance, the average chronaxia of the deltoid is .10 millisecond, and it is .30 for the extensor digitorum communis. This test appears to be of great practical value in the study of neuromuscular degeneration. In injuries of the lower motor neurons, the chronaxia increases with the degree of the injury.

After severance of a motor nerve, the muscle being completely denervated, the normal and rapid contraction elicited by electrical stimulation is replaced by a slow wormlike response. The chronaxia of all the fibers composing such a muscle may increase to as much as 120 times its normal figure.

The diagnosis and prognosis of lesions of the lower motor neurons are aided by this test. Neuromuscular degeneration can be demonstrated the first week after the onset of the paralysis. It becomes very evident after three weeks, because it takes some time for the process to reach its full extent. In a paralysis resulting from lesions of the upper motor neurons, or in muscles atrophied from disuse, chronaxia changes practically not at all.

The activity of nerves and muscles is accompanied by action potentials. A motor-nerve fiber and the group of muscle fibers supplied by it is called a motor unit. Action potentials represent discharges from motor units. They can be recorded with an oscillograph. In these experiments, an ink-writing oscillograph was used. Six records could be taken simultaneously from protagonist and antagonist muscles. Small electrodes were applied to the skin with paste; coaxial pairs of needle electrodes were also sometimes used.[3,4]

EXPERIMENTAL RESULTS

Fifty-one patients were tested. The time elapsed between the onset of the disease and the tests varied from ten days to one year. Muscles called "alienated" as well as muscles in "spasm" were explored, and muscles considered to be normal clinically were also tested. In all the paretic or paralyzed muscles called "alienated", there was evidence of neuromuscular degeneration. Among these muscles some were in total neuromuscular degeneration (completely denervated). These muscles showed the characteristic wormlike response, and failed to respond to the stimulation of their motor nerves. The chronaxias of these muscles often reached more than 100 times their normal figures. Most of the "alienated" muscles were found in a state of partial degeneration (incompletely denervated). Such a muscle could partially respond to stimulation of its motor nerve; the amplitude of this response obviously depends upon the number of muscle fibers escaping denervation. The chronaxia measured at the motor point was increased, and reached about twenty to fifty times the normal figures. In patients tested one year after the onset of the

paralysis, some muscles classified as "alienated" seemed to have completely disappeared; no contraction could be elicited, even when very strong currents were applied.

Paralysis and neuromuscular degeneration are generally found in certain muscles or groups of muscles. These muscles are, in order of frequency: in the upper extremity, the deltoid, biceps, brachialis, brachioradialis, pronator teres, opponens pollicis, infraspinatus, supraspinatus, and the clavicular part of the pectoralis major; in the trunk, the rectus and oblique abdominis, mostly in their lower parts; in the lower extremity, the tibialis anterior, quadriceps, extensor digitorum brevis, extensor hallucis longus, extensor digitorum longus, and the peronei. The hamstring and the calf muscles were also affected, but less frequently.

Muscles in so-called "spasm" were also tested. "Spasm" is an ill-defined symptom; it is generally used by Kenny to express a certain degree of painful reflex shortening of a muscle. It is said to be sometimes located by observation or gentle palpations of the muscle, but it is usually better demonstrated when the muscle is passively stretched. With the patient at rest, "spasm" is supposed to cause its tendon to stand out prominently. "Spasm" is said to be frequently found, for instance, in the hamstring muscle, but in many normal subjects the tendons of these muscles are very apparent, and it is not easy to tell whether they are more prominent than normally. Furthermore, the normal tension or tonus of any muscle escaping degeneration might simulate "spasm". Finally, the stretching of some totally paralyzed muscle may sometimes elicit pain without "spasm". Therefore, the only means for detecting with accuracy the reflex contraction called "spasm" is by the study of action potentials.

Action potentials were recorded in sixteen patients. Where "spasm" was present, elicited usually by the stretching of the muscle, electrical activity was recorded. Artefacts due to voluntary movements were carefully avoided. In all patients, whether or not the legs were involved, "spasm" was recorded in the hamstring muscles, when these muscles were passively stretched. "Spasm" was recorded in some patients in the gastrocnemius, but very rarely in the extensor digitorum longus, tibialis anterior, or quadriceps. In the adductors of the thigh and in the tensor fasciae latae, "spasm" could not be recorded. In the upper extremity, activity produced by the stretching of the muscles was found less frequently. It was sometimes found in the biceps, in the extensor digitorum communis, very seldom in the flexor digitorum sublimis, and in none of the cases in the pectoralis. As a rule, "spasm" could not be detected in any of these patients at rest. It was always found that the more marked the degeneration, the less extensive was the "spasm" (Fig. 1).

A few examples will illustrate these findings.

Case 1, B. C., a seventeen-year-old male, was tested six and one-half months after the onset of poliomyelitis. Table I represents only part of the chronaxia measurements.* The biceps, deltoid, brachioradialis, and extensor digitorum communis were "alienated" according to Kenny's concept. They showed marked neuromuscular degeneration (chronaxia figures ranged between about fifty and 100 times their normal value, and contraction was slow); the anterior and medial portions of the deltoid and the extensor digitorum communis did not respond to stimulation of their motor nerves. The flexor digitorum sublimis, triceps, trapezius, and pectoralis had approximately normal chronaxia, and there was no evidence of degeneration; these muscles were in so-called "spasm".

Case 2, A. Z., an eleven-year-old male, was examined one year after the onset of poliomyelitis. There was a paresis of the left lower leg and also an incomplete paralysis of the abdominal muscles, evidenced clinically by difficulty in rising from the lying into the sitting position, and prominence of the abdominal wall in the erect position, mostly in its lower part. Table II represents only part of the test.*

There was a partial neuromuscular degeneration in the tibialis anterior and in the exten-

* Cases 1 and 2 were tested in Minneapolis. The author wishes to thank Miland E. Knapp, M.D., for his permission to publish these cases.

TABLE I

CASE B. C.

Muscle or Nerve Tested	Chronaxia (*In Milliseconds*)	Normal Chronaxia (*In Milliseconds*)	Quality of Contraction	Condition of Muscle by Kenny Concept
Left				
Biceps	11.6	.08 to .16	Slow	"Alienated"
Deltoid,—anterior	12.0	.08 to .16	Slow	"Alienated"
Deltoid,—medial	12.0	.08 to .16	Slow	"Alienated"
Deltoid,—posterior	6.0	.08 to .16	Slow	"Alienated"
Circumflex (response in posterior deltoid only)	.3	.08 to .16	Rapid	
Coracobrachialis	10.0	.08 to .16	Slow	"Alienated"
Brachioradialis	14.8	.08 to .16	Slow	"Alienated"
Extensor digitorum communis	18.0	.16 to .32	Slow	"Alienated"
Flexor digitorum sublimis	.6	.16 to .32	Rapid	"Spasm"
Median nerve	.4	.16 to .32	Rapid	
Radial nerve (response in triceps only)	.3	.16 to .32	Rapid	
Triceps	.36	.16 to .32	Rapid	"Spasm"
Pectoralis major,—lower portion	.36	.08 to .16	Rapid	"Spasm"
Trapezius,—upper portion	.2	.08 to .16	Rapid	"Spasm"

TABLE II

CASE A. Z.

Muscle or Nerve Tested	Chronaxia (*In Milliseconds*)	Normal Chronaxia (*In Milliseconds*)	Quality of Contraction	Condition of Muscle by Kenny Concept
Left				
Tibialis anterior	9.2	.16 to .32	Slow	"Alienated"
Extensor hallucis longus	4.0	.16 to .32	Slow	"Alienated"
Extensor digitorum longus	6.0	.16 to .32	Slow	"Alienated"
Extensor digitorum brevis	.4	.16 to .32	Rapid	
Peroneus longus	.28	.16 to .32	Rapid	"Spasm"
Peroneus brevis	.52	.16 to .32	Rapid	"Spasm"
Peroneal nerve	.4	.16 to .32	Rapid	
Gastrocnemius	.32	.16 to .32	Rapid	"Spasm"
Tibial nerve	.44	.16 to .32	Rapid	
Rectus abdominis,—upper portion	.26	.08 to .16	Rapid	"Alienated"
Rectus abdominis,—lower portion	No response	.08 to .16		
External oblique,—upper portion	.24	.08 to .16	Rapid	"Alienated"
External oblique,—lower portion	No response	.08 to .16		
Right				
Rectus abdominis,—upper portion	.12	.08 to .16	Rapid	"Alienated"
Rectus abdominis,—lower portion	No response	.08 to .16		
External oblique,—upper portion	.08	.08 to .16	Rapid	"Alienated"
External oblique,—lower portion	No response	.08 to .16		

sors of the toes. These muscles were in a state corresponding to the "alienation" of the Kenny doctrine, and the calf muscles opposing them were under some tension,—therefore, by definition, in "spasm". The calf muscles had normal function and normal chronaxia.

In the lower part of the abdominal muscles—rectus abdominis and external oblique—no contraction could be elicited even with a very strong current. The anterior-horn cells

TABLE III
CASE D. C.

Muscle or Nerve Tested	Chronaxia (*In Milliseconds*)	Normal Chronaxia (*In Milliseconds*)	Quality of Contraction	Condition of Muscle by Kenny Concept
Right				
Deltoid,—anterior	18.0	.06 to .16	Slow	"Alienated"
Deltoid,—medial	16.0	.06 to .16	Slow	"Alienated"
Deltoid,—posterior	10.4	.06 to .16	Slow	"Alienated"
Circumflex nerve (partial response in the posterior portion of the deltoid)	.16	.06 to .16	Rapid	
Biceps	12.0	.06 to .16	Slow	"Alienated"
Brachioradialis	1.2	.06 to .16	Rapid	
Pectoralis major	.52	.06 to .16	Rapid	"Spasm"
Triceps,—long head	10.0	.16 to .32	Slow	
Anconeus	10.0	.16 to .32	Slow	
Extensor digitorum communis	.68	.16 to .32	Rapid	
Flexor digitorum brevis	.2	.16 to .32	Rapid	
Abductor minimi digiti	2.0	.16 to .32	Rapid	
Opponens pollicis	16.0	.16 to .32	Slow	
Infraspinatus	5.2	.16 to .32	Slow	
Teres major	.4	.06 to .16	Rapid	
Sternocleidomastoideus	.12	.06 to .16	Rapid	
Trapezius,—upper portion	.16	.06 to .16	Rapid	"Spasm"
Left				
Deltoid,—anterior	9.2	.06 to .16	Slow	"Alienated"
Deltoid,—medial	6.6	.06 to .16	Slow	"Alienated"
Deltoid,—posterior	8.0	.06 to .16	Slow	"Alienated"
Circumflex nerve	.16	.06 to .16	Rapid	
Biceps	14.0	.06 to .16	Slow	"Alienated"
Brachioradialis	1.0	.06 to .16	Rapid	
Triceps,—long head	.8	.16 to .32	Rapid	
Triceps lateralis	7.2	.16 to .32	Slow	
Pectoralis major	.3	.06 to .16	Rapid	"Spasm"
Trapezius,—upper portion	.14	.06 to .16	Rapid	"Spasm"

of the tenth, eleventh, and twelfth thoracic segments, and, probably also, of the first lumbar segment seemed to have been completely destroyed. The upper portion of the same muscles had normal excitability. The abdominal muscles were "alienated" according to Kenny's concept.

Case 3, D. C., a twelve-year-old girl, was tested two months after the onset of poliomyelitis.

In the right upper extremity the deltoid, biceps, triceps, anconeus, and opponens pollicis were in marked neuromuscular degeneration. Contraction elicited by galvanic stimulation was sluggish, and the chronaxia reached about 100 times the normal figures. The brachioradialis, abductor minimi digiti and infraspinatus were also in degeneration, but less affected. All these muscles were so-called "alienated", according to Kenny's concept. In the left upper extremity, the deltoid, biceps, and part of the triceps were in degeneration, although less marked than the corresponding muscles of the right side. No trace of degeneration was found in the trapezius and pectoralis major on both sides. These normal muscles were in "spasm" according to Kenny's criteria; they are supposed to be the affected ones.

Case 4, M. J., a boy, six and one-half years old, had meningeal signs, as usually seen in poliomyelitis, four months prior to the electrical test. These were stiffness of the neck.

TABLE IV
CASE M. J.

Muscle or Nerve Tested	Chronaxia (*In Milliseconds*)	Normal Chronaxia (*In Milliseconds*)	Quality of Contraction	Condition of Muscle by Kenny Concept
Right				
Tibialis anterior	.20	.16 to .32	Rapid	
Extensor digitorum longus	.28	.16 to .32	Rapid	
Peroneus longus	.60	.16 to .32	Rapid	
Peroneus brevis	.16	.16 to .32	Rapid	
Gastrocnemius	.28	.16 to .32	Rapid	
Biceps femoris	.32	.16 to .32	Rapid	"Spasm"
Semitendinosus	.28	.16 to .32	Rapid	"Spasm"
Quadriceps	.16	.06 to .16	Rapid	
Left				
Tibialis anterior	.20	.16 to .32	Rapid	
Extensor digitorum longus	.28	.16 to .32	Rapid	
Peroneus longus	.28	.16 to .32	Rapid	
Peroneus brevis	.28	.16 to .32	Rapid	
Gastrocnemius	.24	.16 to .32	Rapid	
Biceps femoris	.28	.16 to .32	Rapid	"Spasm"
Semitendinosus	.40	.16 to .32	Rapid	"Spasm"
Quadriceps	.16	.06 to .16	Rapid	
Frontalis	.24	.16 to .32	Rapid	
Orbicularis oculi superior	.48	.16 to .32	Rapid	
Orbicularis oculi inferior	.40	.16 to .32	Rapid	
Orbicularis oris superior	8.0	.16 to .32	Slow	
Orbicularis oris inferior	6.0	.16 to .32	Slow	
Quadratus labii inferioris	.40	.16 to .32	Rapid	
Facial nerve	.32	.16 to .32	Rapid	

back, and hamstring muscles,—so-called "spasm" according to Kenny's concept. At that time, a left facial paralysis developed. There was no paresis or paralysis in any of the other muscles of the body, and at no time was "spasm" found in the facial muscles. Electrical tests showed facial paralysis on the left side. Neuromuscular degeneration was found in the orbicularis oris, superior and inferior (Table IV). Chronaxias of the muscles of the lower extremities were within the normal range and no trace of degeneration was found in these muscles. On stretching of the hamstring muscles of either leg, pain was present, and action potentials of high amplitude were recorded. According to Kenny's criteria, "spasm" was still present. This case showed that in spite of the so-called "spasm", no "alienation" could be claimed at any time in the muscles of the limbs.

Case 5, E. S., a five-year-old girl, was tested four months after the onset of poliomyelitis. Electrical stimulation of the left lower extremity evidenced marked and extensive injuries to the anterior-horn cells. In some muscles the chronaxias were greatly increased, and reached more than 100 times their normal figures (Table V). The contraction of these muscles was slow. Muscles supplied by the peroneal nerve, except the peroneus brevis, were in total degeneration and did not respond to stimulation of the motor nerve. The gastrocnemius, and the hamstring muscles, as well as the quadriceps, were also in degeneration. On the right side there was no paralysis or paresis,—the figures of chronaxia were close to their normal range, and there was no evidence of neuromuscular degeneration.

With passive straight raising of either leg to an angle of 50 degrees, pain was elicited, and thus, by definition, the muscles were in so-called "spasm". As seen in Figure 1, the activity recorded from the affected hamstring (left) was much smaller when compared with the normal side. Thus, in comparing activities recorded from the hamstring muscles

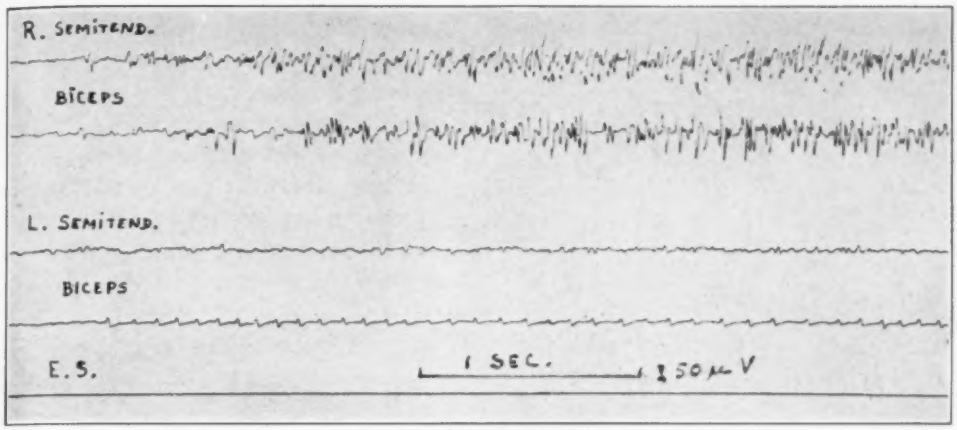


FIG. 1

on both sides, it was found that the greater the degeneration, the less extensive was the so-called "spasm". Furthermore, in spite of the marked "spasm" of the right hamstring

TABLE V
CASE E. S.

Muscle or Nerve Tested	Chronaxia (*In Milliseconds*)	Normal Chronaxia (*In Milliseconds*)	Quality of Contraction	Condition of Muscle by Kenny Concept
Left				
Tibialis anterior	22.0	.16 to .32	Slow	"Alienated"
Extensor digitorum longus	20.8	.16 to .32	Slow	"Alienated"
Extensor digitorum brevis	16.0	.16 to .32	Slow	"Alienated"
Peroneus brevis	4.0	.16 to .32	Slow	
Peroneus longus	16.0	.16 to .32	Slow	
Extensor hallucis longus	26.0	.16 to .32	Slow	
Peroneal nerve (response in peroneus brevis only)	.8	.16 to .32	Rapid	
Gastrocnemius	8.0	.16 to .32	Slow	
Vastus lateralis	4.8	.06 to .16	Slow	"Alienated"
Rectus femoris	20.0	.06 to .16	Slow	"Alienated"
Femoral nerve (slight response in lateral portion of the quadriceps)	.3	.06 to .16	Rapid	
Biceps femoris	10.4	.16 to .32	Slow	"Spasm"
Semitendinosus	16.0	.16 to .32	Slow	"Spasm"
Right				
Tibialis anterior	.24	.16 to .32	Rapid	
Extensor digitorum longus	.8	.16 to .32	Rapid	
Extensor digitorum brevis	.4	.16 to .32	Rapid	
Peroneus longus	.28	.16 to .32	Rapid	
Peroneus brevis	.28	.16 to .32	Rapid	
Peroneal nerve (response in all the muscles supplied by it)	.48	.16 to .32	Rapid	
Gastrocnemius	.3	.16 to .32	Rapid	"Spasm"
Vastus lateralis	.28	.06 to .16	Rapid	
Rectus femoris	.4	.06 to .16	Rapid	
Femoral nerve (response in every portion of the quadriceps)	.3	.06 to .16	Rapid	
Biceps femoris	.38	.16 to .32	Rapid	"Spasm"
Semitendinosus	.32	.16 to .32	Rapid	"Spasm"

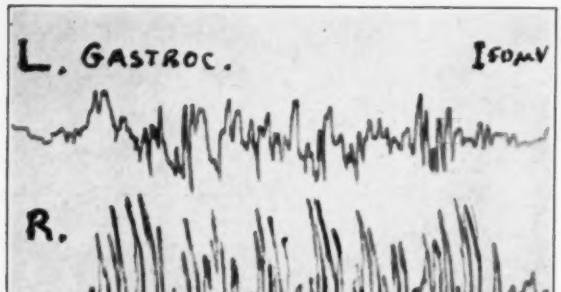


FIG. 2

muscles (normal leg) there was no so-called "alienation" of the quadriceps, simply because there was no neuromuscular degeneration in this muscle.

The stretch reflex was also studied in patients with peripheral nerve injuries, in patients with spastic paralysis, and in normal subjects.

Case 6, H. M., a forty-year-old male, had an injury of the right peroneal nerve which had been accidentally severed one year prior to the examination. Paralysis of the muscle supplied by this nerve was complete. Chronaxia was increased to about 100 times the normal figures, as is often found in complete denervation.

Activities of the gastrocnemius were recorded (Fig. 2). On passive dorsiflexion of the right foot to an angle of 90 degrees, action potentials of high voltage were recorded from the gastrocnemius of the affected leg; they were obviously of greater amplitude than those recorded from the non-affected leg.

This example showed the normal increase of the stretch reflex in antagonist muscles unopposed by completely paralyzed protagonists.

Case 7, T. O., a man, forty-two years old, had a left spastic paralysis due to thrombosis of the branches of the right middle cerebral artery. This patient had typical pyramidal signs, hyporeflexia, knee and ankle clonus, and Hoffmann and Babinski signs on the left side. On passive dorsiflexion of the foot, discharges from the motor units were no longer asynchronous in character as in the normal stretch reflex. The synchronous discharges represented a series of jerks (clonus) as shown in Figure 3.

This example was taken in order to show a clonus, which is one of the characteristic symptoms found in a true spastic paralysis.

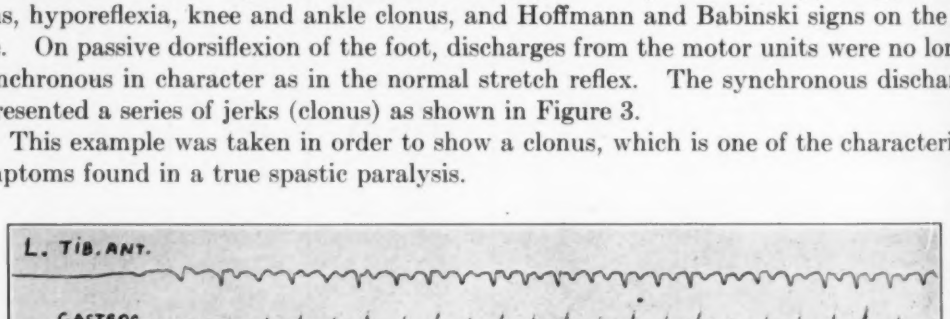


FIG. 3

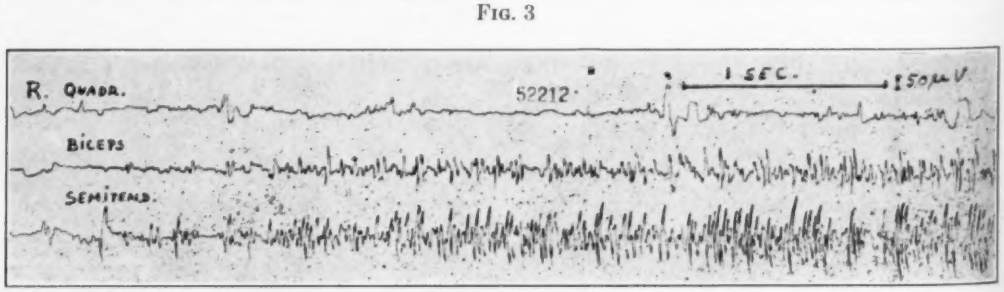


FIG. 4

Case 8, F. C., a twenty-year-old male, was a normal subject. Passive raising of the right leg to an angle of 60 degrees elicited some uncomfortable tension in the hamstring muscles.

In the stretched hamstring muscles, action potentials of high voltage were recorded (Fig. 4). They were in this case as high, if not higher, than in any of the poliomyelitic patients tested.

DISCUSSION

Poliomyelitis is primarily a disease of the central nervous system; all skeletal muscle changes are secondary. The principal and most crippling symptoms are muscle paralysis and paresis. There are also some prominent early symptoms, apparently due to irritation of the meninges and inflammation of the sensory protoneurons.

According to Kenny's concept, "alienated" muscles are muscles which have lost their ability to produce voluntary movements. These muscles are erased from the patient's mind because their antagonists are in "spasm". No mental condition or any "physiological block" is needed to explain "alienated" muscles. Paralysis of a muscle in poliomyelitis is always accompanied by some degree of neuromuscular degeneration; this is the result of damage to the anterior-horn cells. Degeneration will be more or less marked, depending upon the intensity and extent of the lesions. If the chronaxia is slightly increased (partial neuromuscular degeneration), then there is a good chance that the involved muscles may recover completely. This fact explains why some so-called "alienated muscles" regain their normal strength. Some muscles, also considered to be "alienated" by definition, were found in total neuromuscular degeneration, thus indicating that all the fibers composing the muscle were in deep degeneration. The chronaxia was markedly increased. These muscles are no longer able to respond to stimulation of their motor nerve. In infantile paralysis, muscles with total degeneration are doomed, because their loss of function is the result of destruction of all the corresponding anterior-horn cells. A muscle is generally supplied by motor neurons from several segments of the spinal cord. Therefore, total degeneration, implying a more widespread lesion, is not so frequent as partial degeneration. Other muscles, also considered to be "alienated" according to the Kenny criteria, seemed to have completely disappeared and were probably replaced by fibrous tissue. In these muscles, the proliferation of fat and connective tissue had masked the atrophy.

In several cases, the clavicular part of the pectoralis major was denervated, while the sternal part had normal chronaxia; yet the entire muscle had been diagnosed in "spasm". In this instance, the degeneration was as marked in the clavicular part of the pectoralis major as it was in the corresponding deltoid. The reason for this is obvious,—we were dealing with a deep injury in the fifth and sixth cervical segments of the spinal cord, while the lower segments were spared.

In some patients with paralysis of the lower extremities, the lower parts of the abdominal muscles were found in total degeneration. The rectus abdominis, for instance, is supplied mostly by the six lower thoracic segments. Among these segments it can happen that the tenth, eleventh, and twelfth may be completely destroyed, while the upper ones escape degeneration. According to Kenny, "spasm" of the lumbar muscle has "alienated" the abdominal muscles. The rectus abdominis would then be "alienated" in its lower part, and not in its upper part. When the muscles of the thenar eminence are paralyzed, atrophic, and in degeneration, they are also called "alienated". In that case there is never a question of spasm in the abductor pollicis longus or in the extensors of the thumb.

The distinction between a definitely paralyzed muscle and a so-called "alienated" muscle, which might regain some function, thus becomes rather vague. When the injury is very extensive and involves one or two limbs completely, the proponents of the concept

agree to the idea of complete paralysis. When the injury affects a smaller area of the spinal cord and when only a few muscles are paralyzed or paretic, they call these muscles "alienated", while the normal and strong antagonists are said to be in "spasm". It is only a question of degree and extent of the injury, and there is no reason to use a different terminology for the same physiopathological disorder.

The motor deficiencies will be better understood if one bears in mind the pathology and the physiopathology of the disease. In poliomyelitis, one should not consider only whole muscles, but the motor units which compose them. The virus attacks motor cells. Their axons and muscle fibers are secondarily affected. Some motor units may be destroyed, some may be in chromatolysis, and some others will escape damage. A muscle may recover some strength, even when many motor cells supplying it have been destroyed. In the opinion of the writer, some of the transitory paralyses could be explained by the fact that most of the affected motor cells were in chromatolysis without destruction of the corresponding axons. In all these transitory paralyses, the muscle is able to respond to stimulation of its motor nerve. The chronaxia at the motor point of a muscle which is going to recover is increased, and reaches the figures found in a partial degeneration, but never those high figures found in complete denervation.

According to Kenny, muscle "spasm", rather than paralysis, is the primary symptom in poliomyelitis. "The affected muscles are painful, hyperirritable and in spasm" (Pohl and Kenny, p. 38). "Untreated spasm by destroying muscle tissue causes atrophy. If allowed to continue, the final result is frequently a fibrous inextensible non-elastic mass of tissue" (Pohl and Kenny, p. 40). The findings reported here showed that all muscles in marked "spasm" had normal chronaxias,—that is to say, without any trace of neuromuscular degeneration.

As stated previously, the principal location of muscle "spasm" and the most frequent distribution of neuromuscular degeneration make it obvious that "spasm" has nothing in common with denervation and destruction of muscle fibers. "Spasm" consequently is not the "most damaging symptom" of the disease. We know that lesions of the lower motor neurons lead to paralysis, atonia, and abolition of the tendon reflexes, and this is the opposite of "spasm" or hypertonicity. Denervated muscles are frequently in fibrillation, but activity of single fibers cannot give a sustained contraction or tetanus of a muscle and cannot be responsible for "spasm". In an incompletely denervated muscle, some of the motor units escaping damage respond like normal structures. Thus, in a patient with unilateral partial degeneration of the hamstrings and calf muscles, "spasm" might be found on both sides, though always more marked in the normal leg (Fig. 1). "Spasm" is not a separate clinical entity. Under the heading of "spasm" three different symptoms appear to be included. The most important component of the so-called "spasm" is obviously meningeal irritation. At the onset of the disease, the principal location of "spasm" is, roughly speaking, almost the same as the stiffness found in any meningitis,—neck, back, hamstring, and calf muscles. This rigidity of the back muscles in a meningitis has much the same mechanism as that seen in peritoneal inflammation. Here the boardlike reaction, or spasm, of the abdominal muscles is explained by the irritation of the sensory endings of the splanchnic nerves. Stimulation of the central end of the splanchnic nerve in the experimental animal gives rise to a reflex contraction of the abdominal muscles and flexion of the hip. The "doubled-up" attitude seen in cases of peritonitis represents the same reflex mechanism in the abdomen and the lower extremities. An analogous muscle spasm may be found, for instance, at the onset of tuberculosis of the hip joint; it may be so marked that it may simulate ankylosis. It is due to the irritation of the sensory endings in the joint. It has never been suggested that spasm of the muscles in a meningitis, peritonitis, or tuberculosis of the hip joint is a damaging symptom and will lead to destruction of the muscles. In these conditions it is merely a reflex defense phenomenon. In infantile paralysis, some muscles other than those found in meningeal

irritation or in meningitis are found in "spasm". In a meningitis, for instance, tension in the pectoralis major or latissimus dorsi is generally not found. It should be mentioned that, in some severe lesions of the lower motor neurons of different etiology than poliomyelitis, there may be marked shortening of the uninvolved antagonist muscles. For instance, in lesions of the brachial plexus with paralysis of the deltoid, biceps, brachioradialis, coracobrachialis, triceps, and extensors of the fingers, it is not infrequent to find marked shortening, which might be called "spasm", in the normal superior trapezius, pectoralis, and latissimus dorsi. This is a distribution often found in poliomyelitis.

Thus a second type of "spasm", according to the Kenny definition, is sometimes seen in the antagonists of a muscle paralyzed from any cause. In a patient (Case 6) who had the peroneal accidentally severed, the paralysis of the muscles supplied by this nerve was complete, and showed total neuromuscular degeneration. On passive dorsiflexion of the foot, action potentials recorded from calf muscles evidenced an increase of the stretch reflex compared with the normal side (Fig. 2). Thus, in peripheral nerve injuries, a picture similar to the so-called "spasm" was found. The increase of the stretch reflex of protagonist muscles unopposed by completely paralyzed antagonist muscles, in the experimental animal, was described by Sherrington. It is explained by lack of the normal inhibition of protagonist muscles (reciprocal inhibition of Sherrington).

The third factor which might also account for part of the mechanism of "spasm" is the inflammation of the posterior-root ganglia which usually exists in poliomyelitis.[1, 2, 5, 10] "The posterior root ganglia show the same cellular infiltration and cytological changes. Flexner states that experimentally lesions of the ganglia are as common and constant as the lesion of the grey matter, and in man are probably as constant, but not so frequently looked for."[1] "Spasm" is said to be most easily demonstrated by the stretching of the muscles. When a muscle, with its reflex arc intact, is elongated, afferent impulses, originating in the muscle spindles, stimulate the spinal cord; efferent impulses are sent back through the motor nerve and elicit a reflex contraction (stretch reflex of Sherrington). When the sensibility of the posterior root is increased by an inflammatory process, or when the dorsal-root ganglia are irritated, the threshold of the reflex can be lowered, and the response is stronger. These afferent impulses passing through affected ganglia and inflamed roots will produce a referred pain in the muscles. Pain due to stretching of the muscles can be explained by a mechanism of summation of impulses, because the stronger the stretch, the greater the pain.

"Spasm" has, therefore, not a single etiology; it may be caused by one, two, or even all of the three factors described in the previous pages. The meningeal component of "spasm" disappears relatively quickly. This meningeal reaction explains why it is more marked at the onset of the disease. Irritation of the posterior horns and ganglia may last longer than meningeal reaction. The last and the most persistent type of "spasm" is that due to the increase of the normal tonus in strong muscles.

Besides these three factors, it should be mentioned that, in a normal subject, the stretch reflex of the hamstring and calf muscles was sometimes as marked as in any case of poliomyelitis. In normal subjects, action potentials of high amplitude were recorded (Fig. 4).

The third symptom described is "incoordination". According to Kenny, it is due to a "disorganization of the regulating centers".

In infantile paralysis, the voluntary and the automatic control of movement is impaired only because nerve impulses are directed to normal as well as to completely and incompletely denervated muscles. This is seen in any case of injury of the lower motor neuron. It is true for lesions of the anterior-horn cells and for injuries of the peripheral nerves.

If among the muscles which have to carry out motion, some are paralyzed or paretic, the patient will attempt to substitute a stronger muscle for a paralyzed one. This leads

to an abnormal and unusual use of the extremity. An example for this will be a case in which the tibialis anterior is completely denervated, the extensors of the toes are partially denervated, and the peronei are normal. If the patient is asked to dorsiflex his foot, the motion will consist of a slight dorsiflexion combined with an abduction resulting from the stronger action of the peronei. The term "incoordination", in the sense commonly used in neurology, does not apply to this type of substitution. Kenny also uses the term "incoordination" when a partially paralyzed muscle contracts in one part, but not in the other. For instance, when the different portions of the quadriceps are involved, but less in its lateral portion, the latter might, in that case, be the only one able to respond in a voluntary effort to contract the quadriceps. This too has been frequently recognized, but it certainly does not warrant the term "incoordination".

In the usual cases of poliomyelitis, there is neither change in deeper sensibility nor are there lesions in the cerebellum which might lead to what is called clinically "incoordination".

SUMMARY

The chief disorders resulting from infantile paralysis have been considered in this paper. The principal and most crippling symptoms are muscle paralysis and paresis. This is the result of lesions of the anterior-horn cells. In the affected muscles, neuromuscular degeneration can be accurately determined by chronaxia. The degree and the extent of the injury are evaluated by this test; this aids in the diagnosis and the prognosis. Inflammation of the sensory protoneurons is not necessarily found at exactly the same level as the injuries to the anterior-horn cells. Therefore, pain and tenderness of muscles are found in non-paralyzed as well as in affected muscles. Pain might be explained as a referred pain which is usually increased by the stretching of the muscles.

The Kenny concept is fundamentally different. Three cardinal symptoms are described,—muscle "spasm", "mental alienation", and "incoordination". To test this concept, a series of investigations have been carried out, since obviously a clear understanding of the symptoms is of great importance for the diagnosis, the prognosis, and the treatment of any disease. The various parts of the concept have been considered.

1. "Spasm" is not "the most damaging symptom of the disease"; it is a complex and ill-defined phenomenon, under which several different conditions are included. "Spasm" does not lead to neuromuscular degeneration.

2. In paralytic and paretic muscles called "alienated", our observations showed that there was always some degree of neuromuscular degeneration. Among these muscles, some were partially denervated; these have a good chance to recover. Some others were totally denervated, and, therefore, will not recover.

3. There is no clinical evidence of "incoordination" in the ordinary sense. The patient attempts voluntarily or involuntarily to use a stronger muscle for a weak or paralyzed one. This abnormal use of an extremity is substitution and not "incoordination".

The author is indebted to Tracy J. Putnam, M.D., for his help and interest in this work. He wishes to thank Paul F. A. Hoefer, M.D., for the use of the electromyographic equipment, and Alan DeF. Smith, M.D., and William R. Snow, M.D., for referring some of the patients.

Most of these patients were tested at the Neurological Institute of New York; twelve patients were tested at the Elizabeth Kenny Institute, Minneapolis, and twelve at the University Hospital, Minneapolis.

REFERENCES

1. Batten, F. E.: Acute Poliomyelitis. Brain, XXXIX, 115, 1916.
2. Flexner, S.; Clark, P. F.; and Amoss, H. L.: A Contribution to the Pathology of Epidemic Poliomyelitis. J. Exper. Med., XIX, 205, 1914.
3. Hoefer, P. F. A.: Innervation and "Tonus" of Striated Muscle in Man. Arch. Neurol. and Psychiat., XLVI, 947, 1941.

4. Hoefer, P. F. A., and Putnam, T. J.: Action Potentials of Muscles in Normal Subjects. Arch. Neurol. and Psychiat., XLII, 201, 1939.
5. Hurst, E. W.: The Histology of Experimental Poliomyelitis. J. Pathol. and Bacteriol., XXXII, 457, 1929.
6. Kenny, Elizabeth: The Treatment of Infantile Paralysis in the Acute Stage. St. Paul, Bruce Publishing Co., 1941.
7. Moldaver, J.: Physiopathologic Aspect of the Disorders of Muscles in Infantile Paralysis. Preliminary Report. J. Am. Med. Assn., CXXIII, 74, 1943.
8. Pohl, J. F., and Kenny, Elizabeth: The Kenny Concept of Infantile Paralysis and Its Treatment. St. Paul, Bruce Publishing Co., 1943.
9. Sherrington, C. S.: Quantitative Management of Contraction in Lowest Level Co-Ordination. Hughlings Jackson Lecture. Brain, LIV, 1, 1931.
10. Warburg, Bettina: Experimental Poliomyelitis. Histology of the Persistent Lesions of the Central Nervous System. Arch. Neurol. and Psychiat., XXV, 1191, 1931.

DISCUSSION

Dr. Alan DeF. Smith, New York, N. Y.: I might explain to you that Dr. Moldaver has been in this country about two and one-half years. He was interested originally in neurologic disease. He has been working at the Neurological Institute in New York, and has examined these cases of anterior poliomyelitis under a grant from The National Foundation for Infantile Paralysis. In addition to his experience in this country in examining cases treated by the Sister Kenny method, he has a background of several thousand cases examined in Belgium.

We who know about the pathology of poliomyelitis are not surprised at his results. They confirm what we have been taught, and they disprove the concept of the disease put forth by Sister Kenny. I do not think anyone who has tried that method has agreed with her concept of the disease.

There is another question as to whether, regardless of the fact that her ideas are wrong, the treatment has any value. In the summer of 1941 we began to treat a group of cases at the New York Orthopaedic Hospital and we have treated 100 or more patients. The treatment has been used for too short a time to give a final report. We have endeavored to carry out this treatment exactly as Sister Kenny described. At first there was difficulty, because there was no publication that gave it accurately, but Sister Kenny came and gave us some suggestions. When the course was started at the University of Minnesota, two of our physiotherapists and two of our nurses went there and took the course; so I believe we have carried out the treatment as it should have been done. Most of our cases were mild cases, and we would expect those patients to recover with a small amount of residual deformity, with almost any treatment. That has happened. We have had some severe cases with an extreme degree of paralysis of the trunk muscles and lower extremities. I do not think those cases have any less degree of paralysis following treatment by the Kenny method than they would have had by any other method. I believe that, if the anterior-horn cells are destroyed, the muscle is permanently paralyzed, and you cannot expect any good result. I believe there has been less contracture of the joints and less stiffness of the muscles after this treatment than by our former methods of treatment. In the past, we probably erred by immobilizing the patients too much and too long. I think this method has helped to rectify that.

Spasm has persisted for a long time. We have had cases in which there has been so-called spasm a year after treatment. I think too much attention has been paid to spasm, and I believe that it does less harm than it is supposed to. I think some people have been treating spasm, rather than the paralysis. Hot packs do not always relieve the spasm, because it is still present after many months of treatment in some cases. We have used braces where we thought it necessary. The only ill effect I saw after using this method was that, in four or five cases, we have had contracture of the calf muscles where the feet have not been supported. They have been allowed to assume a position of equinus, according to Sister Kenny's statement that you must not do anything to the muscles in spasm; in those cases we have recently started to use supports.

I would like to speak about a paper that was circulated privately. One of our residents, who had had no previous experience with this method, compared a group of mild cases treated by the Sister Kenny method with a group of cases treated by the conservative method, and published a paper. I do not want you to think that that paper represents the ideas of the New York Orthopaedic Hospital concerning the Kenny method.

Dr. Leo Mayer, New York, N. Y.: I want to stress the importance of Dr. Moldaver's paper. For eighteen years he has been doing the chronaxia reaction and may well, therefore, be considered an expert in this particular field. His methods are scientific; he is quite objective, since he has no axe to grind. His conclusions, after testing a considerable number of recent poliomyelitis cases, are that the Kenny concept is entirely wrong. His work did not deal with the Kenny method of treatment, but with the fundamental question of the Kenny concept of infantile paralysis. This is the particular contribution which Sister Kenny herself stresses as the important thing; she claims that she has established a new concept of infantile paralysis. To prove whether

this is right or wrong, we require some accurate method of testing the function of the neuromuscular apparatus. This can be done accurately by means of the chronaxia reaction which indicates with precision whether the muscle responds normally to its nerve supply. Sister Kenny, as you know, claims that the muscles in spasm are those affected by the virus of the disease, whereas those that we formerly considered paralyzed are only temporarily "alienated" because of the "spasm" of their opponents. Dr. Moldaver has shown that this idea is completely wrong; the "spastic" muscles are the normal muscles with normal chronaxia reaction; the alienated muscles show marked changes in the chronaxia, indicating that the nerve supply has suffered serious damage. In other words, the muscles which we formerly considered paralyzed are the paralyzed muscles, and we are, therefore, not in as complete ignorance of the disease as Sister Kenny would make us out.

I, for one, am most grateful to Dr. Moldaver for this paper. I am sure that when you have a chance to read it, you will understand many details which were not entirely clear at the time of its presentation.

Dr. R. Plato Schwartz, Rochester, New York: Dr. Ransohoff and Dr. Moldaver have stated their respective views regarding the Kenny treatment and concept of infantile paralysis. I wish to express personal accord with the objective attitude which these authors clearly revealed. It is both wholesome and essential for unbiased conclusions regarding this subject at the present time. The claims made in support of the Kenny treatment are based entirely on clinical observation. On this same clinical basis, the value of the Kenny treatment is still subject to question for at least two good reasons: (1) This method of treatment has not been used long enough in relation to the known variables of the disease to have end results for comparison with those from other methods of treatment, which point was emphasized by Dr. Ransohoff; (2) during the past three decades in particular, definite and specific knowledge of the pathology of infantile paralysis has been acquired. Recent investigations confirm observations made thirty years ago. As yet there is no correlation between the pathology thus established and the Kenny approach to the solution of this problem.

This brings me to the consideration of Dr. Moldaver's paper. He has emphasized the dangers associated with the Kenny concept that "spasm is ever-present and damaging" to the restoration of function. By the determination of chronaxia he found that Kenny treatments were being given for relief of spasm, when muscles were completely paralyzed. One year ago we presented evidence to this society indicating the presence of spasm as determined by action currents. This investigation of the reaction of muscles during the acute stage of infantile paralysis has been continued during the past year. This work has confirmed previous findings.

But we must not misinterpret the significance of this evidence of muscle spasm during the acute stage of infantile paralysis. The established pathology of the disease remains unaltered. The reasons are obvious. Muscles which reveal no evidence of weakness, weakened muscles, and the antagonists of weakened muscles all have revealed evidence of spasm. Where clinical evidence of muscle spasm is greatest, as in the neck and back (spine sign), there is a much lower incidence of disability than in the extremities.

As stated by Dr. Smith, we likewise have found no correlation between the use of hot packs and the diminution of spasm. The duration has varied between a few days and fifteen months. Evidence to date supports the conviction that, like fever and other clinical manifestations of infantile paralysis, muscle spasm in variable degree runs its course independent of hot packs and without a revealed relationship to the disabilities which may prevail in the chronic stage.

Dr. Joseph A. Freiberg, Cincinnati, Ohio: I have enjoyed Dr. Moldaver's paper tremendously and much that I wish to say Dr. Schwartz has said already. I have had the pleasure of working with Dr. Albert Sabin for a period of time in Cincinnati. He is now in Service. He is a real authority on the pathology of infantile paralysis, having done more complete autopsies on patients dying from anterior poliomyelitis than many of us. He has always said that he never changed his views that the pathological findings were as emphasized by Dr. Moldaver. I think that some of us have, on occasion, accepted the clinical explanation for what occurs, actually not based on a true pathologico-physiological basis. The explanation of why certain things happen, because they seem to happen, by one who is not a scientist and who is not a doctor, must be accepted with some skepticism. I have followed a rather large series of cases, larger than Dr. Ransohoff's, and feel that at present we can come to no conclusions because of the relatively small series of cases. We have had patients get completely well in the past, and we have patients get completely well now, many of them treated by physiotherapists trained by Sister Kenny. I have followed the patients treated by Sister Kenny for persistent contractures; one notable case of a patient with curvature of the spine and paralysis of both legs she has treated for a long time, but the patient shows no improvement whatsoever.

Dr. Steele F. Stewart, Honolulu, T. H.: I would like to make one or two observations that perhaps may be at variance with what some authors have stated. In some old cases of five years' duration, the application of packs have permitted patients who had flail legs to dispense with braces and walk with less limp than before. I cannot explain this to you. We have had a number of those cases in Honolulu.

We have an epidemic there at the present time in which we are trying to apply the Kenny method as taught by Sister Kenny. We have two Kenny technicians. There is one thing about which I feel very definite. Sister Kenny's refusal to allow her taught technicians to disseminate any knowledge they have acquired, I feel, borders on charlatanism. I think we should do everything we can to inspire experimentation

in infantile paralysis and pass on freely what we learn without reference to The National Foundation or any other organization.

Talking about complete autopsies, I am not sure that there is not pathology in the sensory organs which should be explored. I do not know whether we have gotten to the bottom of it. As someone said in a previous paper, we never look at nerves.

DR. NICHOLAS S. RANSOHOFF, NEW YORK, N. Y.: I want to thank the discussors. One of the things that I had not intended to go into in this question was the problem of pathology in poliomyelitis. I am wondering whether or not the pathology of poliomyeleitis today is not being looked at incompletely, as Dr. Stewart suggests. We know that the anterior-horn cells show vacuolization in the region of the anterior horn. That does not necessarily show us what is going on and what is the effect in the immediate vicinity of the anterior horns of the inflammatory process, which must be a necessary accompaniment of acute poliomyelitis. This whole inflammatory picture must spread up through the spinal cord to some degree and cannot be localized in this one small area. That may be the answer to some of the statements which Dr. Schwartz has so ably made. I still say that the method is under scrutiny, and we shall tell you more about it when we know more about it.

DR. JOSEPH MOLDAVER, NEW YORK, N. Y. (closing): I wish to thank the discussors. In this presentation the chief disorders resulting from infantile paralysis have been discussed. The Kenny concept was investigated and it was found that this concept of poliomyelitis was incorrect. I think that we should be very cautious in evaluating the result of any treatment in a disease in which spontaneous improvement is almost a rule. In order to appreciate the real value of different treatments, cases with the same degree of neuromuscular degeneration should be compared. Clinically speaking, paralyzed muscles might appear involved to the same degree, while electrical tests might show great differences and consequently the prognoses will not be the same. Accurate electrical tests should always be made, if one wants to have an exact evaluation of different treatments. Furthermore, these tests will show when to stop physical treatment and when orthopaedic intervention is advisable.

METAL-BLOCK REPLACEMENT OF BONE DEFICIENCY

A Preliminary Report on an Operative Correction for Genu Recurvatum

BY ROSS SUTHERLAND, M.D., LOS ANGELES, CALIFORNIA, AND
M. JOHN ROWE, JR., M.D., LONG BEACH, CALIFORNIA

From the Tichenor Orthopedic Clinic, Long Beach

One of the difficulties encountered in the surgery of bone has been the compressibility of bone grafts used in weight-bearing areas. The individual physiological variables have made true surgical accuracy almost impossible.

A review of the methods of treatment for paralytic genu recurvatum has shown a great variety of surgical treatments which fall into two major classes. The soft-tissue or check-ligament operations, as exemplified by the methods of Gill and Carrell, have shown the ever-present tendency to late relaxation, with partial loss of correction. The second major group of bone operations includes those using the bone-block principle,—as in Campbell's graft of the patella to the tibia[2] and Mayer's tibial block behind the patella. Later, the osteotomy[3] of the upper tibia below the tibial tubercle was advised. However, Brett's procedure seems most sound in its attempt to restore the physiological bone block of the knee joint to hyperextension. This operation can be done prior to closure of the epiphyses, and the method has been used successfully by the authors[7].

FIG. 1

Diagram shows epiphysiotomy with metal block in the defect. Tibial tubercle and patellar tendon have been reflected.

The study of roentgenograms of the hyperextended knee in paralytic genu recurvatum, particularly in the lateral projection, shows a consistent deviation from the normal; the superior articular surface of the tibia is always inclined forward, and is not perpendicular to the axis of the tibia as in the normal knee. Consequently, correction of the deformity requires return of the articular surface to its normal relationship with the tibia. Since this can be accomplished only by osteotomy, the para-articular incision of the bone and the elevation of the anterior portion of the articular surface seem mandatory. This may be done before closure of the epiphyses, and much deformity can be prevented by correction and utilization of normal growth tendencies.

When bone is used to maintain the correction, the variables mentioned enter the picture. Correction may be complete at the time of surgery, but the grafts may be compressed

FIG. 2

Polished stainless-steel wedges. The medium size is most commonly used.

on weight-bearing, allowing a return of the deformity to some extent. If overcorrection is attempted, it is occasionally possible that the compression will not be sufficient to allow complete extension and consequent stability of the knee. The application of metal in such cases was considered. Early experiments in the measuring of bone growth by the implantation of metal particles in animal bones showed such a procedure to be feasible and without evidence of bone irritation. Metal nails in the femoral neck have been better tolerated, when almost completely surrounded by bone, than have equal amounts of metal when surrounded by soft tissue. Apparently in many instances the soft tissues of the extremities were less tolerant of such metallic foreign material than the bone itself, when the object was completely implanted in the bone.

The procedure of tibial epiphysiotomy for genu recurvatum was modified by the use of metal-block wedges in place of cortical-bone grafts, and the surgical technique was further simplified by reflection of the patellar tendon and its attachment to the tibial tubercle. The tubercle was later replaced and fixed with a metal nail. Such an approach was found to be superior to either the parapatellar or the split-patellar approach.

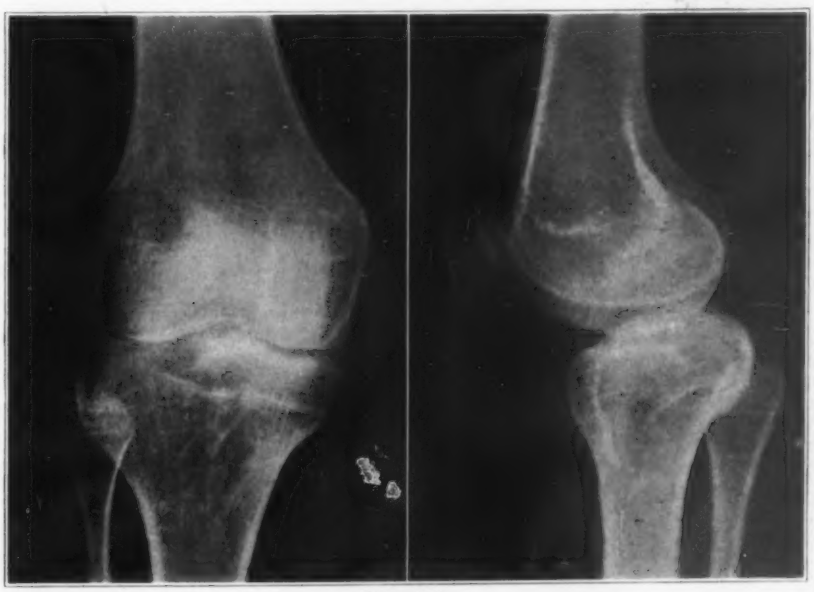


FIG. 3-A

Case 1. C. J. A. Roentgenograms of right knee, before operation, in a patient with bilateral paralytic genu recurvatum.

FIG. 3-B

Roentgenograms of right knee after operation, showing four metal wedges in defect.

TECHNIQUE

A tube tourniquet is applied high in the groin. Through an anterior mid-line incision over the knee, the patellar tendon is reflected at the tubercle by a V cut with a motor saw, and the cut is extended into the joint capsule with a scalpel. The entire knee joint, the superior articular surface of the tibia, and the cartilages are exposed. A transverse linear osteotomy of the tibia, parallel and 0.5 centimeters distal to the articular surface, is begun with the motor saw and finished with a wide thin osteotome. This space is levered apart until the articular surface is at right angles to the shaft of the tibia and the anterior inclination is corrected. A large metal block or wedge of suitable size (Fig. 2) is inserted entirely within the cortical margins, and the correction is tested. The tibial tubercle is then replaced and held with metal nails; the capsule is closed with locked sutures; the remainder of the wound is closed in layers; and a compression bandage is applied from the toes before removal of the tourniquet. No external support is required. Immediate crutch ambulation and joint motion are allowed as tolerated, and weight-bearing is permitted when calcification anterior to the wedge is seen in roentgenograms (usually in from four to six weeks).

FIG. 3-C

FIG. 3-D

Photographs before and after operation for paralytic genu recurvatum of left knee.

The operation is simple and rapid, and correction can be estimated immediately. Hospitalization is short, with early ambulation and function. The correction is positive, because the block is non-compressible.

FIG. 3-E

Roentgenograms of left knee, same individual, before operation.

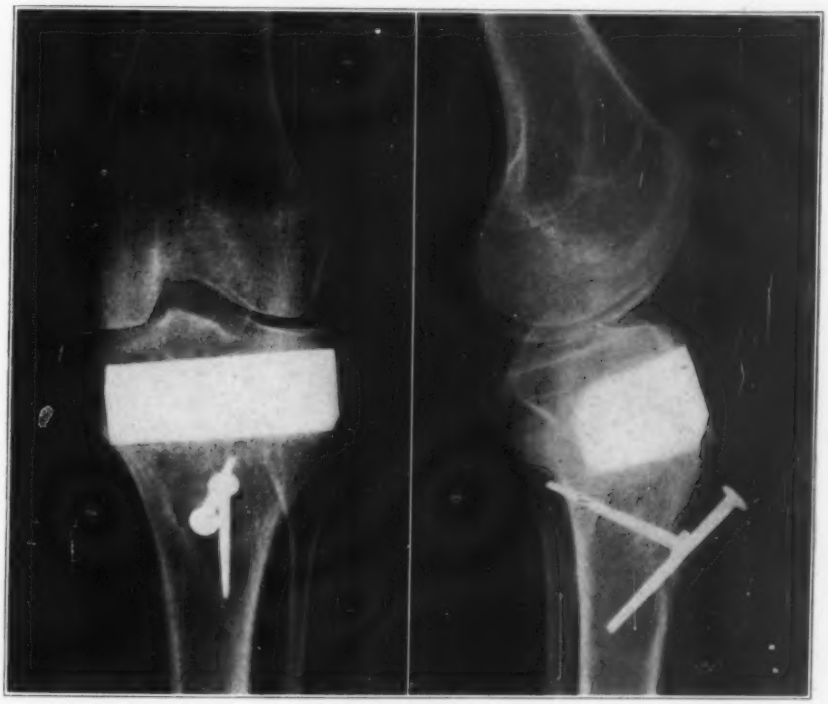


FIG. 3-F

Roentgenograms of left knee after operation, showing wedge in defect. Note correction of anterior inclination of tibial articular surface to bring it perpendicular to axis of tibia.

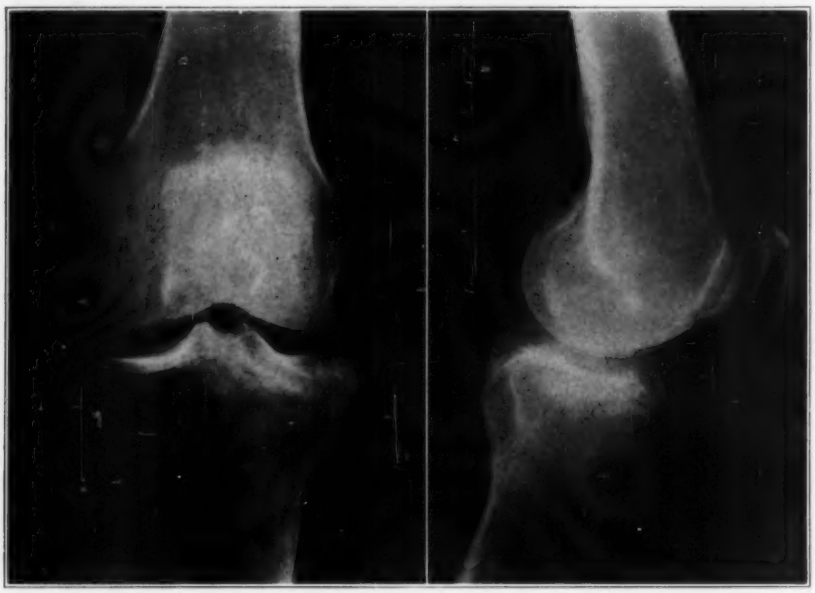


FIG. 4-A

Case 2. A. G. Roentgenograms showing depressed comminuted fracture of the lateral tibial condyle of the left knee.

FIG. 4-B

Roentgenograms of fracture after operation. Elevation has been effected with metal wedges.

CASE REPORTS

CASE 1. C. J. A., a female, aged seventeen years, was admitted with the diagnosis of bilateral paralytic genu recurvatum (Fig. 3-A). She had had poliomyelitis at the age of nine. Subsequent to the attack, ambulation had been possible with bilateral long leg braces and crutches. An epiphysiotomy of the right tibia was performed June 24, 1942. Four stainless-steel wedges were used instead of bone grafts (Fig. 3-B). A long leg cast was applied and worn for two weeks after the operation. She had an uneventful convalescence. Weight-bearing with a brace was allowed four weeks after the operation. The brace was discarded after ten weeks. Her gait was much improved because of increased stability of the knee, and crutches were no longer

FIG. 5-A

Case 3. D. C. Roentgenograms of right knee, before operation, in a patient with paralytic genu recurvatum.

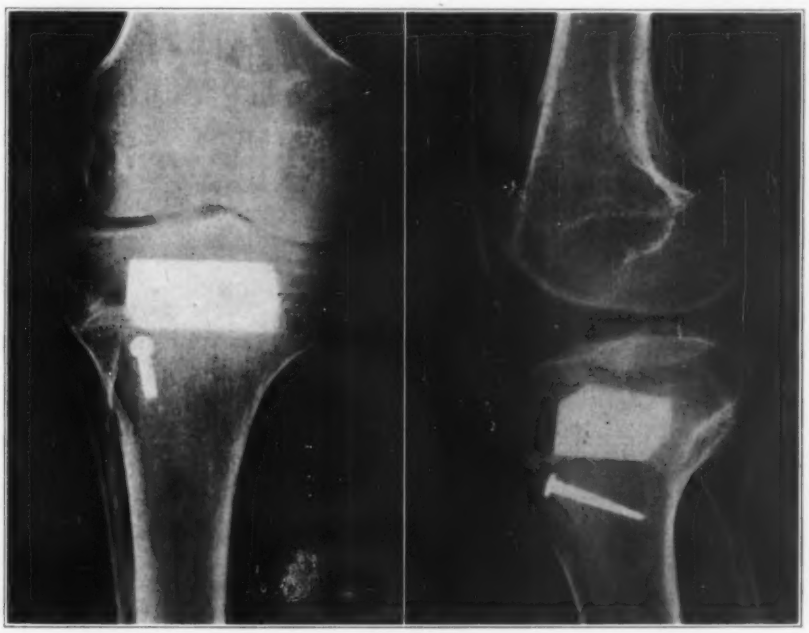


FIG. 5-B

Roentgenograms of right knee after operation, showing metal wedge in position.

required. In March 1943, surgical correction of the left knee was done by the insertion of a single medium-sized steel block. No cast was used. Weight-bearing with crutches was allowed two weeks after the operation. The result was excellent.

CASE 2. A. G., a female, aged sixty-three, had sustained a depressed comminuted fracture of the lateral tibial condyle of the left knee (Fig. 4-A). The injury occurred September 29, 1942. At operation, October 5, 1942, the external cartilage was removed; the condyle was elevated; and the position was maintained with three metal wedges (Fig. 4-B). No cast was used. Motion was allowed as soon as it could be tolerated, and weight-bearing was permitted after four weeks. Five months after the operation, function was excellent.

FIG. 5-C

Photograph of paralytic genu recurvatum before operation.

FIG. 5-D

Photographs of paralytic genu recurvatum after operation.

This case (Case 2) has been included to show the adaptability of the procedure to a traumatic condition such as this one.

CASE 3. D. C., a male, aged eighteen, had a right paralytic genu recurvatum and flail foot (Fig. 5-A). He had had poliomyelitis at the age of twelve. Upon admission he was walking with a long, double upright brace. An epiphysiotomy of the right tibia was performed August 12, 1942, and a stainless-steel metal block was inserted to obtain immediate full correction (Fig. 5-B). A compression bandage was applied without other external immobilization. Ambulation with crutches, and knee motion were allowed in five days, and guarded weight-bearing in a brace was instituted three weeks after the operation. There was no evidence of intolerance of the metal or of delayed healing. Full correction was maintained, and stability improved almost to normal.

SUMMARY

1. Complete exposure of the knee joint is facilitated by the reflection of the tibial tubercle and of the patellar tendon. Early function is instituted, following re-attachment of the tubercle by means of a metal nail.

2. The anterior inclination of the superior articular surface of the tibia must be corrected to relieve the deformity of genu recurvatum and to give adequate knee function.

3. In reconstructive surgical procedures, metal blocks may be used advantageously to fill bone defects.

REFERENCES

1. BRETT, A. L.: Operative Correction of Genu Recurvatum. J. Bone and Joint Surg., XVII, 984, Oct 1935.
2. CAMPBELL, W. C.: An Operation for the Correction and Prevention of Paralytic Genu Recurvatum. J. Am. Med. Assn., LXXI, 967, 1918.
3. CAMPBELL, W. C.: Operative Orthopedics. p. 602. St. Louis, The C. V. Mosby Company, 1939.
4. CARRELL, W. B.: Use of Fascia Lata in Knee-Joint Instability. J. Bone and Joint Surg., XIX, 1018, Oct. 1937.
5. GILL, A. B.: Operation for Correction of Paralytic Genu Recurvatum. J. Bone and Joint Surg., XIII, 49, Jan. 1931.
6. MAYER, LEO: An Operation for the Cure of Paralytic Genu Recurvatum. J. Bone and Joint Surg., XII, 845, Oct. 1930.
7. SUTHERLAND, ROSS, AND ROWE, M. J., JR.: Tibial Epiphysiotomy for Severe Genu Recurvatum. Surgery, X, 927, 1941.

CHIP FUSION OF THE LOW BACK FOLLOWING EXPLORATION OF THE SPINAL CANAL

BY HENRY BRIGGS, M.D., AND PAUL R. MILLIGAN, M.D., EAST ORANGE, NEW JERSEY

There are many reasons, theoretical and technical, that make a fusion desirable following exploration of the low-lumbar spinal canal. The authors wish to describe their method of using bone chips in obtaining such a fusion, and to give a summary of their results.

Theoretically, the object of fusion is to prevent a reherniation of nuclear material, if there has been a removal of a ruptured nucleus pulposus, and to prevent a late constriction of the intervertebral foramen. The intervertebral foramen can be constricted by a compression of the intervertebral space, by an outward bulging of the disc, or by the development of hypertrophic changes. When the mobility and resiliency of the disc is lost, by injury or disease, nature tends to fuse the adjacent bodies by what appears in the roentgenogram as hypertrophic lipping. There will be less inflammation, less calcification, and less constriction of the foramen if a fusion is performed at the time of operation.

Technically, a facet is frequently destroyed or entirely removed at operation, making a fusion desirable to ensure mechanical stability.

The majority of the authors' cases have been explored at the lumbosacral level and a lumbosacral fusion has been performed. When a fourth lumbar disc, or one at a higher level, has been removed, only the adjacent segments involved are now fused. Fusion is carried out from the fourth lumbar vertebra to the sacrum in fifth-lumbar spondylolisthesis, also in fourth-lumbar disc disorders, if the fifth lumbar vertebra seems to be definitely "unstable" at operation.

OPERATIVE TECHNIQUE

The procedure used in a laterally placed, lumbosacral, ruptured disc is as follows:

The patient is anaesthetized, usually in the prone position, and the lumbar spine is flexed by placing pillows under the abdomen. A mid-line incision is made. After reflection of the muscle tissue, the sacrum is carefully cleaned of soft tissue to facilitate the identification of the fifth lumbar segment. This is important. Testing for mobility by leverage on the spinous process is an unreliable guide to the fifth lumbar vertebra, and pathology need not be under the most mobile segment, but may be beneath a segment which is almost fixed. This is so when the whole disc is protruding posteriorly and hypertrophic changes have set in. With bone-cutting forceps, the spinous processes of the fifth lumbar vertebra and the sacrum are removed, and the overhanging distal portion of the fourth lumbar spinous process is chiseled off with an osteotome. Self-retaining retractors are applied. The removal of the spinous processes gives excellent exposure. While the operation is in progress, the nurse clears the processes of soft tissue and breaks them into small chips, roughly three to five millimeters in diameter.

Using a large curette or sprot, and starting laterally, the capsule of the lumbosacral facet on the side of the sciatic pain is stripped off, and then, stroking medially, the ligamentum flavum is removed or turned medially on that side. If, during this process, a contraction of the gluteal muscles on that side is observed, great care must be taken, the ligamentum flavum must be incised medially and reflected laterally, so as not to injure the first sacral nerve root, which is then pressed against the ligamentum flavum. When the ligamentum flavum is broken through, small moist packs cut from dental rolls and threaded with black silk are used to push the epidural veins out of harm's way and to control hemorrhage. The lateral aspect of the interlaminal space is entirely cleared of the attached ligament.

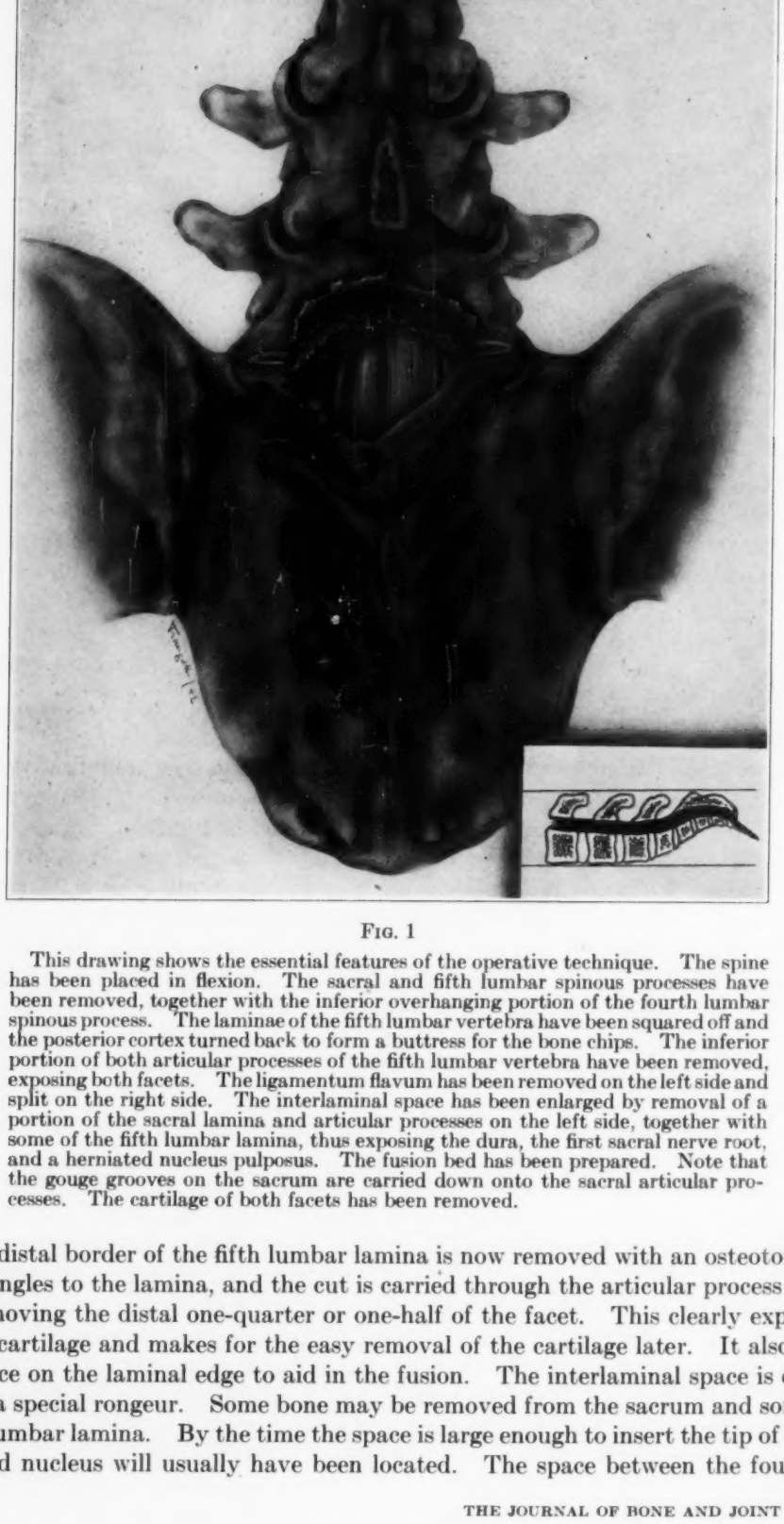


FIG. 1

This drawing shows the essential features of the operative technique. The spine has been placed in flexion. The sacral and fifth lumbar spinous processes have been removed, together with the inferior overhanging portion of the fourth lumbar spinous process. The laminae of the fifth lumbar vertebra have been squared off and the posterior cortex turned back to form a buttress for the bone chips. The inferior portion of both articular processes of the fifth lumbar vertebra have been removed, exposing both facets. The ligamentum flavum has been removed on the left side and split on the right side. The interlaminal space has been enlarged by removal of a portion of the sacral lamina and articular processes on the left side, together with some of the fifth lumbar lamina, thus exposing the dura, the first sacral nerve root, and a herniated nucleus pulposus. The fusion bed has been prepared. Note that the gouge grooves on the sacrum are carried down onto the sacral articular processes. The cartilage of both facets has been removed.

The distal border of the fifth lumbar lamina is now removed with an osteotome, held at right angles to the lamina, and the cut is carried through the articular process into the facet, removing the distal one-quarter or one-half of the facet. This clearly exposes the articular cartilage and makes for the easy removal of the cartilage later. It also gives a raw surface on the laminal edge to aid in the fusion. The interlaminal space is enlarged by using a special rongeur. Some bone may be removed from the sacrum and some from the fifth lumbar lamina. By the time the space is large enough to insert the tip of a finger, a ruptured nucleus will usually have been located. The space between the fourth and

FIG. 2

The nucleus pulposus has been removed, together with as much nuclear material as possible from between the bodies of the vertebrae. A spinous-process peg might have been driven in between the bodies of the vertebrae to stabilize them. The exposed nerve root has been covered by a piece of free fat. The spine has been placed in a normal prone position, as shown in the drawing. The bone chips have been laid in place. On the right side, a squared-off piece of spinous process has been plugged in between the fifth lumbar articular process and the sacrum, and then partially covered by chips. This is an optional variation in the technique, also used to give temporary stability. Note that the bone chips are small.

fifth lumbar laminae may be similarly explored, with the exception that the facet should be preserved unless a decision is later made to include the fourth lumbar segment in the fusion. If nothing is found in either location in this manner, the lumbosacral interspace is explored again, for, in our experience, this is where the pathology usually lies.

The lumbosacral interspace is enlarged laterally by chiseling down the exposed sacral element of the lumbosacral facet and by removing the fragments with a curette. Under the overhang of the facet, which is usually anteroposterior in position, a protrusion impinging the first sacral nerve root is frequently found. We do not hesitate to go farther and remove the entire sacral articular process and enough of the fifth lumbar process to expose the fifth lumbar nerve root in the intervertebral foramen. Failure to explore the intervertebral foramina has allowed lesions to be missed in several cases.

After the ruptured nucleus has been located, the first sacral nerve root and the dura

are retracted medially with a hook. The bleeding is controlled by the threaded packs; the ligamentous tissue is incised when necessary; and the ruptured nucleus is removed with an artery clamp or a nasal forceps. If the whole disc is found bulging posteriorly, a large window is cut in the annulus fibrosus with a knife, and enough of the annulus fibrosus and nuclear material is removed to relieve nerve-root pressure. If this is not enough, the entire facet is excised. As much nuclear material as possible is removed from between the bodies of the vertebrae. We have packed bone chips between the vertebral bodies in an effort to obtain an intercorpus fusion, but without success.

When the nuclear material has been removed, the area is packed with moist packs, and the ligamentum flavum is stripped on the opposite side, splitting it only, and leaving enough to cover the dura. The inferior margin of the fifth lumbar lamina is removed, together with the distal portion of the articular process, as on the other side. The cartilage of both facets is cut away, as completely as possible, with chisel and curettes. The posterior cortices of both fifth lumbar laminae are now turned up with an osteotome to form a retaining buttress for the bone chips. With a gouge, the cortex of the exposed sacrum is removed. Care is taken to obtain raw bone laterally and to carry the gouge grooves from the sacrum down to the exposed sacral articular processes. Any long chips turned up from the sacrum are fragmented.

The packs are now removed. If considerable epidural bleeding remains, it is controlled by clamp and cautery, ligatures, silver clips, or packing. Packing with muscle usually controls it. Moderate venous epidural bleeding is not serious. If the first sacral nerve root lies widely exposed, it is covered by a piece of free fat or muscle.

The elevation under the patient is now removed so that a normal prone lordosis is obtained. (If this is not done before the chips are placed, they will later be displaced upward to some extent.) The chips are next laid in place. The remains of the facets are filled and chips are packed between the facet and the sacrum laterally. The smallest chips are placed over the dura. When all the chips are in place, there will be a mass extending lengthwise from the fifth lumbar lamina with the turned-up buttress, covering the first sacral lamina, and crosswise from facet to facet.

In order that patients may be allowed up after ten days, before fusion can have taken place, and while the possibility of settling of the vertebrae is present, the tip of a spinous process has frequently been squared and plugged in between the fifth lumbar facet and the sacrum, on the opposite side from where the nucleus pulposus has been removed, for increased temporary stability. The technique now in use is to fashion a round peg, and drive it in between the bodies of the vertebrae from which nuclear material has been removed. This gives immediate, demonstrable stability, and is the procedure of choice.

The patient is returned to bed with a simple flat dressing. He is not allowed to turn in bed or to flex the thighs more than is necessary for balance while on the side. Otherwise, he may lie supine, prone, or on either side. When the stitches have been removed, at the end of ten days, a light, low-back brace is applied. With this brace the patient may turn freely, and is allowed up after two weeks. Most patients leave the hospital within three weeks. In cases of spondylolisthesis, patients are kept in bed usually for six weeks. Most patients cling to the brace for some months, to which there is no contra-indication.

DISCUSSION OF CASES

Following the technique described, the spinal canal has been explored and a chip fusion performed in seventy cases in which the senior author was the responsible surgeon. The longest period of follow-up in this series has been thirty-seven months and the shortest, one month (in the most recent case), or an average of fourteen and two-tenths months.

Group I

Thirty-eight of the patients had definite disorders of the intervertebral disc, twenty-

nine having a herniation of the nucleus pulposus, and nine, a posterior protrusion of the intervertebral disc.

Every patient from whom a herniated nucleus was removed has made a satisfactory recovery. In the nine patients in whom a posterior protrusion was found, seven made good recoveries and two made fair recoveries. Of the latter, one had a postoperative infection and the other had an extensive pre-existing hypertrophic arthritis of the lumbar spine. In this group, four facetectomies were performed.

Group II

In three early cases with definite signs of a herniated nucleus pulposus, no disc pathology was found. We now feel that pathology was missed at operation. One patient made a satisfactory recovery, but the results in the other two cases have been poor.

Group III

In seven cases of spondylolisthesis, three hemilaminectomies and four complete fifth-lumbar laminectomies were performed. Additional bone for chips was obtained from an ilium. Recovery was satisfactory, with solid fusion from the fourth lumbar vertebra to the sacrum.

Group IV

There were twenty-two individuals operated upon under the diagnosis of unstable fifth lumbar vertebra.* These patients suffered from low-back pain, usually with radiation of pain to the gluteal areas and the lower extremities. They presented no definite neurological findings. All but two have made satisfactory recovery. One young woman states that she has had no relief from her symptoms, and one man, fifty-five years of age, a compensation case, has never returned to work.

Generally speaking, the results in this group have not been so satisfactory as in the group in which definite disc pathology has been found and removed. This seems to be due to the selection of cases rather than to the operative technique. However, the results are as good, if not better, than the results obtained from a low-back fusion alone. Three of the patients in this group have been operated upon a second time. In none of them was there any evidence of overgrowth of callus pressing on a nerve root. Incidentally, one patient (the compensation case) obtained no relief from the second operation. In one patient, a facet was removed, with an excellent result. The third patient showed failure of fusion from the fourth lumbar vertebra to the sacrum; a fifth-lumbar unilateral facetectomy afforded no relief. This young woman is very active. Objectively she has no disability, but still complains of pain.

SUMMARY AND CONCLUSIONS

From our experience, we feel that a tremendous quantity of bone chips, as is used in cases of spondylolisthesis, can be placed over the dura with impunity, if the nerve roots are covered by muscle or fat. The method is simple and quick, and in most cases it results in solid fusion. It is somewhat difficult to obtain a solid fusion from the fourth lumbar vertebra to the sacrum without taking chips from an ilium.

Formerly, fusion was routinely carried out from the fourth lumbar vertebra to the sacrum, after removal of a ruptured nucleus of the fourth lumbar vertebra; now the fusion is extended only from the fourth to the fifth lumbar vertebra in these cases.

* It is the senior author's theory that most chronic low-back pain is due to muscle spasm protecting a nerve root from injury, and that disc or foraminal pathology is present in these cases. Actually, a loose fifth lumbar vertebra results from a degenerated lumbosacral disc. The diagnosis as used here is merely a classification.

To date, the young woman previously mentioned is the only patient in whom there has been definite evidence of failure of fusion.

The conclusions which we have drawn from this work are that fusion of the adjacent segments should be performed following operation for disorders of the intervertebral disc, and that a chip fusion, as described, is a satisfactory operative procedure.

The authors are indebted to Dr. J. Lawrence Pool, neurosurgeon, for valuable aid in eight of the cases presented. The majority of the operations were performed at the New Jersey Orthopaedic Hospital.

REFERENCES

Chamberlain, W. E., and Young, B. R.: The Diagnosis of Intervertebral Disk Protrusion by Intraspinal Injection of Air. Air Myelography. J. Am. Med. Assn., CXIII, 2022, 1939.

Dandy, W. E.: Recent Advances in the Diagnosis and Treatment of Ruptured Intervertebral Disks. Ann. Surg., CXV, 514, 1942.

Serious Complications of Ruptured Intervertebral Disks. J. Am. Med. Assn., CXIX, 474, 1942.

Duncan, William, and Hoen, T. I.: A New Approach to the Diagnosis of Herniation of the Intervertebral Disc. Surg. Gynec. Obstet., LXXV, 257, 1942.

Hadley, L. A., and Snook, T.: Intervertebral Foramens. (Scientific Exhibit.) Am. Med. Assn., Atlantic City, June 8, 1942.

Hagman, G. L.: Personal Communication.

Love, J. G.: Protruded Intervertebral Disks. With a Note Regarding Hypertrophy of Ligamenta Flava. J. Am. Med. Assn., CXIII, 2029, 1939.

Love, J. G., and Camp, J. D.: Root Pain Resulting from Intraspinal Protrusion of Intervertebral Discs. J. Bone and Joint Surg., XIX, 776, July 1937.

Mauric, G.: Le disque intervertébral: physiologie, pathologie et indications thérapeutiques. Paris, Masson et Cie, 1933.

Mixter, W. J., and Barr, J. S.: Rupture of the Intervertebral Disc with Involvement of the Spinal Canal. New England J. Med., CCXI, 210, 1934.

Spurling, R. G., and Bradford, F. K.: Neurologic Aspects of Herniated Nucleus Pulposus. At the Fourth and Fifth Lumbar Interspaces. J. Am. Med. Assn., CXIII, 2019, 1939.

CARPECTOMY FOR INTRACTABLE FLEXION DEFORMITIES OF THE WRIST*

BY J. WARREN WHITE, M.D., AND SAM G. STUBBINS, M.D., GREENVILLE, SOUTH CAROLINA

From the Shriners' Hospital for Crippled Children, Greenville

Flexion deformities of the wrist, in addition to interfering seriously with the function of that joint as well as the hand, are most unsightly. They are awkward and complicate tremendously the act of dressing and putting the hand in the pocket,—so desirable in these disabled extremities, simply to get them out of the way. Any hitherto unpublished procedure that will produce a better-looking part in the severe cases, almost regardless of the improvement in function, is felt to be worth reporting, particularly after the method of correction has stood the test of time and is well thought of by both the patient and the surgeon.

In the hands of the senior author, the alternative procedures recommended for the correction of this serious condition have met with so little success in the severe cases that he has devised the operation presented here for its correction. Steindler's flexor muscle-stripping operation just distal to the elbow does not give sufficient slack in these seriously fibrosed forearms to permit a desirable amount of correction. The extensive flexor tendon-lengthening operation resulted in such an unwieldy tendinous scar mass that it was abandoned. The bone-shortening procedure is too much surgery for a forearm already badly damaged.

FIG. 1

Photograph of the articulated bones of the wrist after the removal of the carpus, showing the nice anatomical apposition of the radius with the proximal articular surface of the metacarpus, particularly the fitting of the radial styloid into the pocket between the bases of the first and second metacarpals after the removal of the greater multangular.

While no similar procedure has been found described in the literature, in the most recent edition of Campbell's "Operative Orthopedics", the removal of the proximal row of carpal bones has been recommended for moderate deformities of this nature. Apparently the nice anatomical fitting of the base of the metacarpals upon the distal dorsal aspect of the radius was not appreciated, nor was the advantage recognized of getting more slack in the tight flexors by the total resection of the carpus, so necessary in the severe cases. For this reason the procedure lends itself particularly to the extreme deformities where the removal of only half the carpus would not allow full correction of the flexion.

Until one examines for himself the articulating surface of the proximal ends of the metacarpals and the distal end of the radius, he does not fully understand the advantage of this operation. The distal, slightly dorsally slanting surface of the rounded end of the articular surface of the radius is well adapted to the shape of the slightly concave, proximal metacarpal articular surfaces, the radial styloid fitting nicely into the corner made by

* Read at the Annual Meeting of The American Academy of Orthopaedic Surgeons, Chicago, Illinois, January 19, 1943.

the removal of the greater multangular between the base of the first and second metacarpals (Fig. 1).

In doing the first few carpectomies, the greater multangular was not excised, as it was felt that its removal would result in too much instability of the base of the thumb, but it was noted that this tended also to displace the hand too far laterally. After the greater multangular was removed inadvertently upon one occasion, and a more pleasing result was obtained without sacrificing stability, it was removed routinely.

To be sure, the surfaces make a rather indifferent joint at the start, but it is felt that with use, particularly in children for whom this operation has been primarily devised, the surfaces will gradually accommodate themselves to each other. If a painless, efficient joint results from an astragalectomy in the lower extremity, it is much more reasonable to expect a somewhat similar adjustment to take place in a non-weight-bearing articulation. There naturally will be considerable limitation of motion, but, in the partially disabled arm in which this operation is indicated, this limitation will be of little importance as long as the deformity does not recur. In no instance has a painful joint or a recurrence of the deformity resulted. Ten or twenty degrees of motion, frequently more, can be confidently expected.

FIG. 2

AB: Original longitudinal incision.
A'DB'C: Diamond-shaped piece of skin removed for better closure when the wrist is dorsiflexed.

The distal end of the ulna may be excised, if it appears at the operation to be excessively long. Its removal through a longitudinal lateral incision in half of the cases in this series was considered to be desirable, and apparently has had a favorable effect on the final result.

The carpals are removed through a longitudinal dorsal incision centered over them (Fig. 2). The incision is of sufficient length to permit the use of walling-off towels; in an adult it is usually three or four inches long. The tendons are retracted to either side, and the carpus is generously exposed. Little difficulty is ordinarily encountered in removing the carpals in one piece, starting from the ulnar side. The further acute flexing of the already flexed wrist when the metacarpals are exposed will facilitate their removal. The articulating surfaces of the radius and metacarpals are not disturbed. It is necessary for obvious reasons that the dissection be carried out close to the bone, particularly anteriorly. No tendons need be severed, and, after the carpals are excised, the dorsal fascia is not sutured, as after dorsiflexion it is redundant and comes together naturally. The pisiform is not disturbed as it does not form an integral part of the carpus. It is situated anteriorly in another plane, functions as a sesamoid rather than as a supporting structure, and articulates with only the posteriorly located cuneiform. The operator will be gratified with the nicety of the fitting of the metacarpal bases on the distal dorsal surface of the radius.

A tourniquet is used to facilitate the dissecting, but is released before closure of the wound to permit identification and any necessary ligation of articular vessels which, when present, are mostly the immediate branches from the closely located radial artery. In most of the cases, however, no ligation of any other than the superficial vessels is necessary.

In closing the wound, there is always so much excess skin present on the dorsum of the now dorsiflexed wrist that it is usually desirable to remove a triangular-shaped piece of skin on either side of the original longitudinal incision, making finally a square or diamond-shaped hiatus on the dorsum of the wrist before dorsiflexion (Fig. 2). As the wrist is dorsiflexed, when the metacarpals are fitted on the dorsal edge of the distal end of the radius, the extremities of the original longitudinal incision come closer together, and the new edges of the transverse wound can be sutured, making a much better closure and avoiding an ugly "dog-eared deformity" (Fig. 3). No circulatory difficulty has ever been noted as a result of eliminating the large veins on the dorsum of the wrist.

FIG. 3

Showing "dog-eared" appearance of the ends of transverse incision six months after operation, if suggestions about making the incisions are not followed.

A well-padded, but snugly fitted, plaster-of-Paris dressing is applied, holding the wrist in 20 or 30 degrees of dorsiflexion, and the forearm in mid-rotation, the most useful position, as forearm rotation is markedly limited. For the first three weeks or so the plaster is extended to the axilla, with the elbow at a right angle. Then, if thought desirable, a short skin plaster only to the elbow is employed for another fortnight or so, after which home physical therapy is started. All possible finger action is started early after the operation.

It is surprising to note that in a few months' time, a gratifying amount of dorsiflexion sometimes develops after the correction of the severe flexion deformity, even in severe cases where there appeared to be no trace of this muscle action before the operation.

As one should expect, after noting the direction of the newly approximated articular surfaces, there is usually a moderate amount of ulnar deviation. This, however, is not serious, and does not detract from the generally satisfactory appearance of the hand. There usually persists only a few degrees of motion—just enough to preserve stability and avoid the rigidity of an ankylosed wrist. Ten to twenty degrees of motion dorsally from a position of complete extension, or probably a little hyperextension, is what may be expected. There of course is little or no abduction or adduction (Figs. 4-A, 4-B, 4-C, and 4-D).

With the exception of one case where proximal dorsal displacement of the metacarpals occurred in a congenital club hand (Case 3), the results have been good, and the operation is heartily recommended in severe permanent flexion contractures of the wrist.

It must be remembered, however, that this operation has been devised for the correction of severe deformities not responding to more conservative measures. Any improvement in function of the fingers is incidental, and the primary purpose of the procedure is to improve the appearance of the extremity and to facilitate its being cared for passively, such as in dressing, protection from weather, *et cetera*. One of the chief complaints of these patients has been their inability to keep the ischaemic hand warm.

Twelve wrists in all have been operated upon, the first one five years ago, the others at more or less regular intervals, the last one a few months ago. Four of them were on arthrogrypotic individuals; one on a colored boy with suppurative arthritis of the wrist; the others for ischaemic contractures after severe elbow or severe forearm fractures. No

patients with spastic paralysis were included in this series, as the operation was deemed unsuited to a wrist where the constant muscle imbalance, which it is felt would persist after the operation, would cause a recurrence. The ages of the patients ranged between ten and eighteen, the average age being thirteen. Only two were girls, which is to be expected in a deformity of this nature resulting from severe trauma. The authors are sufficiently enthusiastic about the operation to use it on a suitable adult patient.

Although the final arc of motion in these altered, grossly abnormal joints is seriously limited, it is painless for any activity for which such a deformed member would be employed, and is sufficiently stable for relatively strenuous use, either pushing or pulling, with the usually flexed hooklike fingers.

In conclusion, after operating on these twelve almost hopelessly deformed wrists, the authors feel justified in recommending this operation, devised primarily as a practical way of lengthening the flexor tendons and shortened capsular structures when dorsiflexion cannot be obtained by more conservative means. The less involved dorsiflexors of the wrists have in this series accommodated themselves to this shortened condition of the forearm, and, if not too much damaged, have regained a properly functioning tone. If they do not improve, no harm has been done; and the anatomical structure of the newly formed joint maintains a satisfactory position.

CASE REPORTS

Case 1. J. S., a fifteen-year-old boy, had a carpectomy for ischaemic contracture of the left forearm resulting from a badly fractured elbow and forearm eight years before. Flexion deformity was present

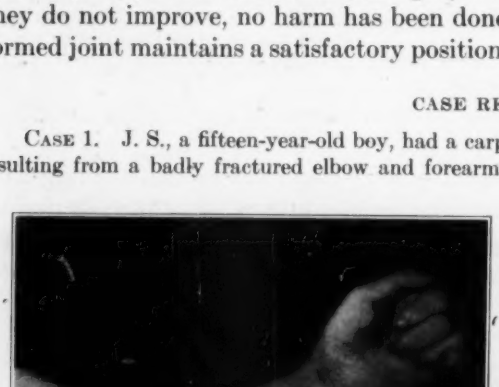


Fig. 4-A

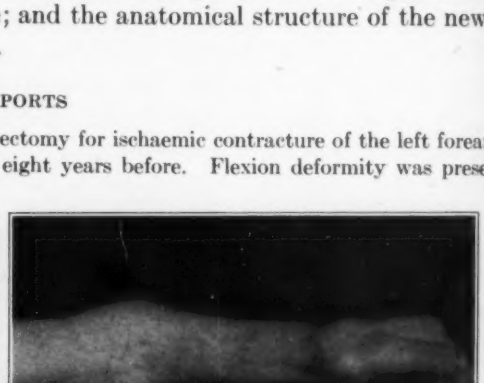


Fig. 4-B

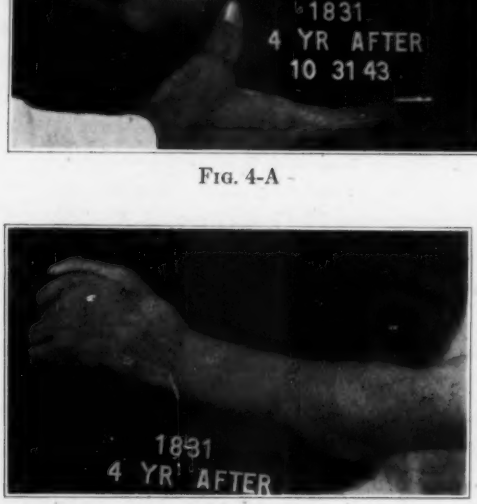


Fig. 4-C

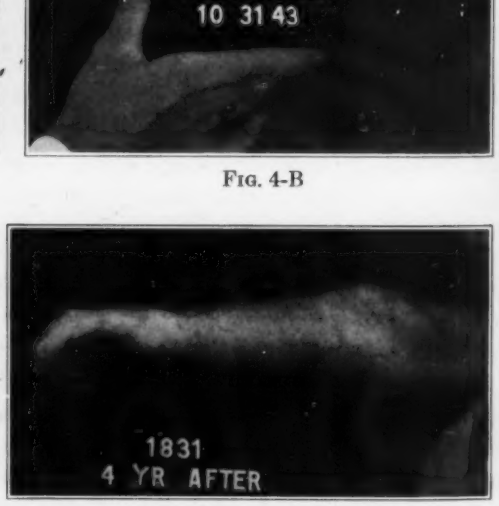


Fig. 4-D

Case 1. J. S. Four views taken four years after carpectomy, showing the typical appearance except that usually there is as much ulnar deviation as radial deviation in such a case, where the distal end of the ulna was not excised. This patient had about 10 degrees of active dorsiflexion, but practically no motion in the fingers. There was some active motion of the thumb, enough to hold a paper and to steady the hand against a specially devised ploughshare, thus enabling him to carry on as a regular farm hand.

to 60 degrees. The operation was performed six years after an unsuccessful transplantation of the forearm tendon. The greater multangular was left in, but the distal end of the ulna was excised. The result over four years later is a hand, with fingers flexed so as to serve as a hook, held in a few degrees of hyperextension, and having about 10 degrees of voluntary motion. There is practically no motion in the hand, but the boy, now nineteen years old, carries on regular farm work. His only complaint is that the hand becomes cold easily when exposed. The extreme shortness of the forearm is typical in early cases of ischaemic contractures. (See Figures 4-A, 4-B, 4-C, and 4-D.)

CASE 2. G. A. had a carpectomy at the age of eleven years. Severe ischaemic contracture of the left forearm, developing after fracture of the left elbow complicated by vascular damage three years before, resulted in a 20-degree flexion deformity of the wrist. Finger extension was possible only from full flexion to one-half full extension. Operation included removal of the greater multangular, but the distal end of the ulna was not excised. The result three years later was a painless wrist, not hyperextendible, but with about 20 degrees of controllable motion in flexion, and sufficient motion in the flexed fingers to hold a knife fairly well (Figs. 5-A and 5-B).

CASE 3. R. S. had a carpectomy at the age of nine years. The operation was performed for a congenital left club hand associated with multiple arthrogrypotic deformities. Incomplete carpectomy was done because of the author's inability to definitely identify the carpometacarpal junction as a result of partial ankylosis, but enough was removed to get good dorsiflexion of the wrist. The end result one and one-half years later was hardly satisfactory, no final note being made on wrist flexion. The patient was unable to use crutches because of other severe deformities, but the senior author recalls that the hand was in only a fair functioning position. Final roentgenograms show 20 degrees of dorsiflexion in spite of dorsal displacement of the metacarpals. There was so much else wrong with this child that little reference was made to the wrist.

FIG. 5-A

Case 2. G. A. Roentgenogram of a "carpectomized" wrist with good function and appearance three years after the operation. This shows the advantage of removing the distal end of the ulna. The shadow of the pisiform may be seen laterally, but this bone is really a sesamoid and does not constitute an integral part of the carpus. Note the nice fit of the radial styloid in the space between the bases of the first and second metacarpals.

CASE 4. C. K. was another patient with arthrogryposis involving the spine, hips, knees, feet, elbows, and wrists. The right wrist was

FIG. 5-B

Showing motion of the wrist which had been "carpectomized" three years ago. There is good motion of the thumb, but the motion of the fingers is only from full flexion to one-half of full extension. The forearm is weak, but there is fair grasping power. Much more motion was present in the fingers in this case than in most of this series. Twenty degrees of permanent flexion was present in the wrist before operation.

carpectomized when the patient was fourteen years old. The entire carpus was removed, which gave such a satisfactory result that, nearly two years later, the left carpus was removed at the same time that an osteoclasis of the knees was done. For some reason the metacarpals of the left hand became dislocated on the distal end of the radius, resulting in a good deal of overriding of the bone ends, but producing a stable wrist which could have been used with crutches if the rest of his deformities had allowed. A little flexion deformity recurred with only a few degrees of motion. The results obtained in this case of arthrogryposis were felt not to have been worth while, although the appearance of the hands was improved.

FIG. 6-A

FIG. 6-B

FIG. 6-C

FIG. 6-D

FIGS. 6-A, 6-B, 6-C, 6-D

Case 6. J. F. H. Lateral, dorsal, and palmar views of the hand and wrist after removal of the carpus, except for the pisiform, and the distal inch or so of the ulna five years previously. Active motion of the wrist was present from 10 degrees above to 10 degrees below full extension. There was very little motion in the flexed fingers, but enough motion in the thumb to grasp a knife or a hammer, as is seen in Fig. 6-D.

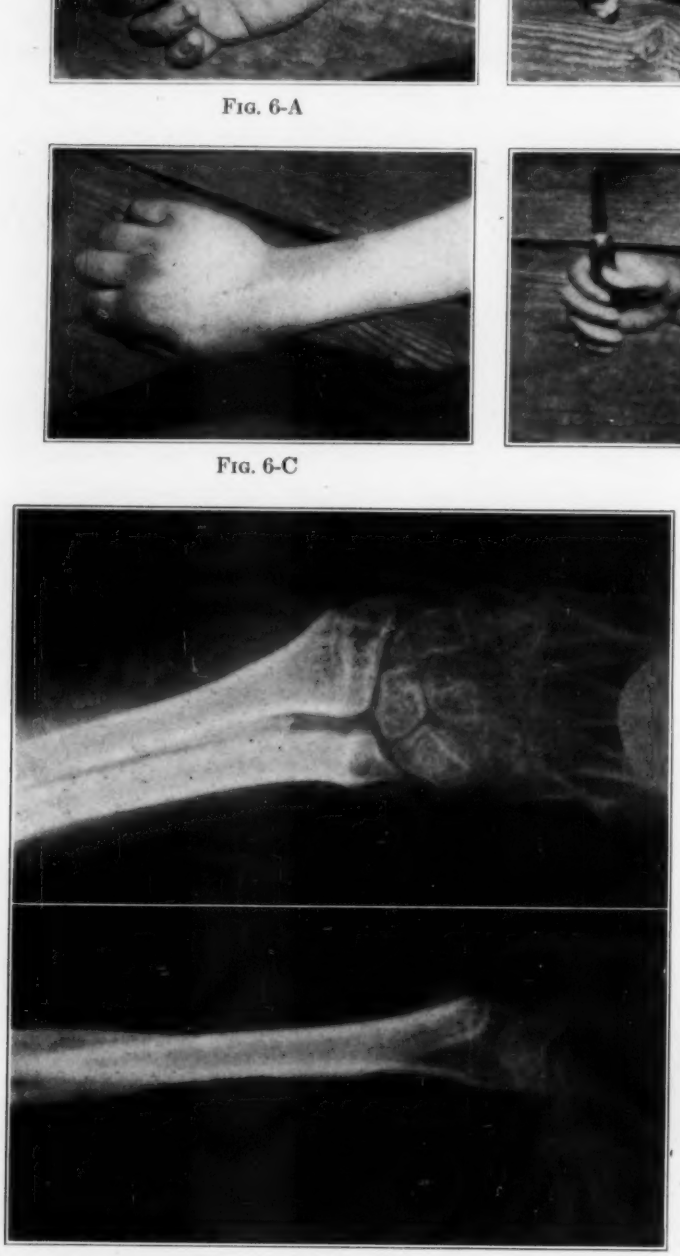


FIG. 7-A

FIG. 7-A

Case 7. F. G. Roentgenogram showing flexion contracture of 70 degrees after an unsuccessful bone-shortening operation on the distal half of the forearm. This is evidence that the long-time contracture of the anterior capsule of the wrist plays a part in maintaining the deformity when it is treated conservatively.

CASE 5. B. B. was nine years old at the time of carpectomy, which was necessitated by a Volkmann's contracture of the right forearm resulting from a severe fracture of both bones in the middle third. A flexion deformity of 60 degrees was present. The greater multangular and distal end of the ulna were not excised, but when the patient was last seen, over two years later, he had over 30 degrees of motion in the wrist in flexion from a few degrees of hyperextension, and full use of the fingers,—sufficient to handle an axe fairly well. This was considered a very satisfactory result.

CASE 6. J. F. H. was nine years old at the time of carpectomy for an ischaemic contracture of the right forearm resulting from a compound fracture of the distal humerus sustained over a year previously. The greater multangular, as well as the distal end of the ulna, was removed. The flexion contracture at the time of operation was 45 degrees with a like amount of a right-angle motion, and very little motion in the fingers. When last seen, over four years later, the patient had very good use of his hand, with controlled motions of wrist about 10 degrees either side of complete extension, and the ability to hold a knife and hammer (Figs. 6-A, 6-B, 6-C and 6-D).

CASE 7. F. G. was thirteen years of age at the time a carpectomy was performed, five years after an unsuccessful attempt at correcting the wrist-flexion deformity of 20 degrees by a shortening osteotomy. However, in spite of the previous osteotomy, flexion deformity of the wrist of 70 degrees had developed (Fig. 7-A). Complete carpectomy was performed without excising the distal end of the ulna. He has not been seen since his last visit seven months after the operation, at which time a desirable amount of dorsiflexion was being maintained (Fig. 7-B). There was very little motion in the hand, but it could be used as a hook.

CASE 8. J. N. was nine years of age at the time of carpectomy which was necessitated by a Volkmann's contracture resulting from a severe compound fracture of both of the bones of the left forearm in the middle third. Two-thirds of the shaft of the ulna had sequestrated, and the radial head had been dislocated and displaced posteriorly. The wrist was flexed to 60 degrees, and there was practically no motion in the fingers, the tendons of which were adherent to the dense forearm cicatrix. The sequestrated ulnar shaft was replaced with a stainless-steel bar, one-quarter of an inch thick, for nine months to prevent further shortening of the forearm. All sinuses healed promptly, and the carpectomy was performed uneventfully six months after the sequestrectomy, good dorsiflexion being easily obtained (Fig. 8). The steel bar was replaced by a tibial graft three months after the carpectomy. Ten months after the carpectomy, she had about 20 degrees of active dorsiflexion from a fully extended wrist, and about half the normal motion of the fingers from a position of full flexion. The forearm was in neutral position, and normal motion of the elbow was present. The child was admitted for an excision of the posteriorly displaced radial head.

FIG. 7-B

Seven months after total carpectomy, during which the distal end of the ulna was not excised, showing how well the metacarpals hold their position on the distal dorsal prominence of the radius.

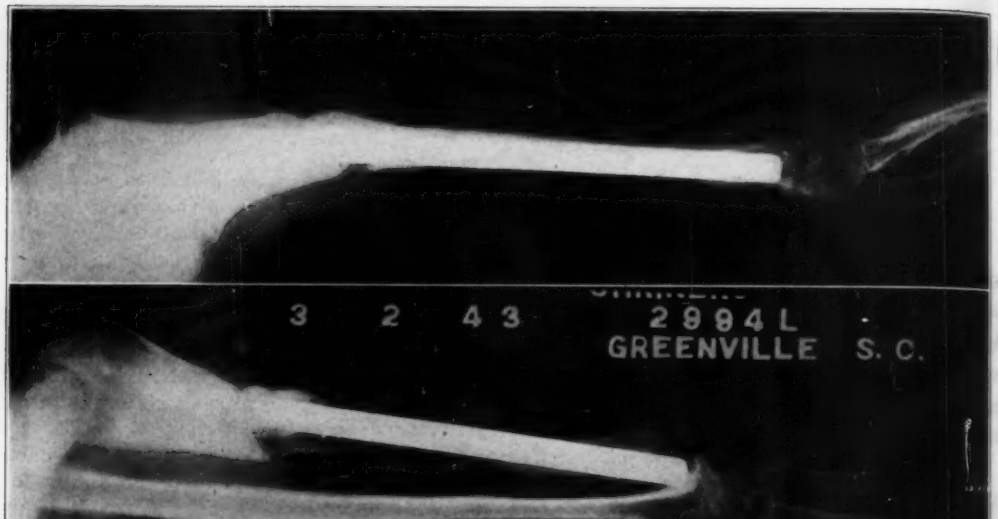


FIG. 8

Case 8. J. N. Roentgenogram taken three months after carpectomy performed for severe Volkmann's contracture with a permanent flexion of 60 degrees below full extension, caused by a compound fracture of the proximal half of the ulna a year previously. The ulnar shaft had sequestrated and a quarter-inch bar of stainless steel had been used to replace it. After this sequestrectomy, all sinuses promptly healed so that a carpectomy could be done six months later. Three months after the carpectomy, the steel bar, having been in for nine months, was successfully replaced by a tibial graft.

CASE 9. J. A., a colored boy, was fourteen years of age at the time of the carpectomy, which was necessitated by a 30-degree flexion deformity resulting from an acute suppurative arthritis of the wrist three years previously. He had practically no motion of that joint or the fingers. The entire forearm was densely sclerosed. The carpectomy was difficult, as the joint lines were hard to identify because of the damage done by the healed suppurative process. Follow-up on this case has been unsuccessful, as the family has moved to an unknown address.

CASE 10. J. P. was nine years old at the date of the total carpectomy with excision of the distal end of the ulna, on January 29, 1941. He had a flexion deformity amounting to 30 degrees, with 20 degrees of further flexion, which resulted from a supracondylar fracture of the left elbow, with serious original displacement, and a simple fracture of both bones of the same forearm two inches proximal to the wrist, also with considerable displacement. Only a few degrees of motion of the thumb and fingers was possible; the fingers were in about two-thirds complete flexion,—enough to use the hand as a hook. The patient was last seen January 25, 1943, two years after the operation, when he had a good-looking hand, capable of picking up something between his thumb and the base of the index finger, and flexed fingers capable of holding a bag, also painless motion of the wrist from 10 degrees flexion to 20 degrees hyperextension—a little more than usual. His mother was much pleased with the result.

CASE 11. A. H. was eighteen years old at the time of carpectomy for a 30-degree flexion deformity of her left wrist,—a rigid congenital deformity associated with severe club feet and a mild similar deformity in her other hand. The operation was a very difficult one, because of the rigidity of all the tissues, and considerable circulatory disturbance developed postoperatively. The entire carpus was removed, but the distal end of the ulna did not have to be removed as it was not in the way. The patient was last seen two and one-half years later when she had good motion in the fingers and motion from about 20 degrees of flexion to a neutral position; hyperextension was possible to 5 degrees. The patient was still suffering some discomfort following an injury to this wrist some weeks previously.

REFERENCES

CAMPBELL, W. C.: Operative Orthopedics, p. 801. St. Louis, The C. V. Mosby Co., 1939.
STEINDLER, ARTHUR: Operative Orthopedics, p. 30. New York, D. Appleton & Co., 1925.

COMPRESSION FRACTURES OF THE VERTEBRAL BODIES AND OTHER CHANGES MISTAKEN FOR THEM *

BY JOHN D. ELLIS, M.D., F.A.C.S., CHICAGO, ILLINOIS

The mechanics of the application of the vectors of force compressing a vertebral body are probably principally dependent upon: (1) the integrity and anatomy of the arches, which, if they are not also destroyed, furnish the fulcrum through which compressive force is applied; (2) the direction and configuration of the articulations of the arches determining the amount of motion occurring at this fulcrum; and (3) the violence of the compressive force applied to the resilient nucleus pulposus and the direction of the dissipation of this force.

It serves no purpose to discuss compression fractures resulting in complete disintegration of a vertebral body and perhaps the adjacent arches, since differential diagnosis of such injuries deserve no detailed description and presents no difficulty. The entire problem of diagnosis of compression fractures of the vertebrae is directed toward those minor fractures which may be simulated by non-traumatic conditions.

A forward-bending force is transmitted without dissipation to the thoracolumbar junction, and it is at this level that the compression fracture most frequently occurs. The point of greatest pressure on any single vertebra comes in the upper lumbar region. Here the intervertebral spaces become wider, and the amount of movement of the body of each vertebra on the one below is greater because of the size of the nuclei pulposi separating the bodies. To a great degree, the freedom of movement is probably also the result of the direction and arrangement of the articular facets of the lumbar spine. Immediately, as one passes down from the thoracic to the lumbar spine, the modal type of lumbar articulation is established. It is only in the lumbosacral joints that variations from this are frequent. The first few degrees of motion in flexion are accomplished through an axis near the center of the nucleus pulposus; and, after the resilience of the nucleus pulposus is exhausted, the fulcrum is established in the joints of the arches. This fulcrum of motion in the intervertebral joints of the arches accounts for the wedge-shaped deformity of a compression fracture in this region, involving, except in the extensive compressions, only the upper half of the affected vertebral body, and principally the anterior upper portion, so that the lateral view shows the typical wedged-deformity compression, the forward tilting of the upper surface often representing one limb of the arc of motion with its angle at the intervertebral joint (Figs. 1-A and 1-B).

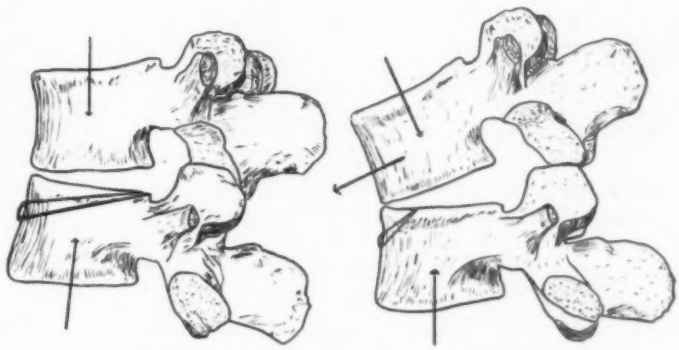


FIG. 1-A

FIG. 1-B

Fig. 1-A: The diagram shows the direction of force producing the typical wedge-shaped compression fracture of the upper surface of a vertebral body.

Fig. 1-B: The diagram shows that the small joints of the arches would be widely avulsed to produce a fracture of the anterior edge of the marginal ring only.

The modal type of lumbar-arch articulations, except the fifth, is in the sagittal plane.

* Read at the Fourth Annual Session of the American Association for the Surgery of Trauma, Boston, Massachusetts, June 6, 1942.

The surfaces of these articulations are slightly curved, so that they are, in effect, sections of a large cylindrical surface, each one slightly narrower at the inferior border. They have been described by Lange as representing the shape of a segment from the neck of a very large bottle in relation to the segment of the adjacent cork. This curve of the joints is not enough to allow any appreciable rotary motion in the lumbar spine at any one level.

However, there is a minimum of definite superior-inferior motion possible at each level, which absorbs a part of the compressive force, and prevents fracture of the arch in a lumbar vertebra, even when the body is badly compressed. In addition to this, there is considerable possibility of forward tilting of each set of zygapophyses on the ones below, so that the upper surface of the fractured vertebra sustains the shearing force of forward movement, as well as an impression force or compression applied in an arc, and therefore the anterior portion of the terminal plate suffers more compression than the posterior portion.

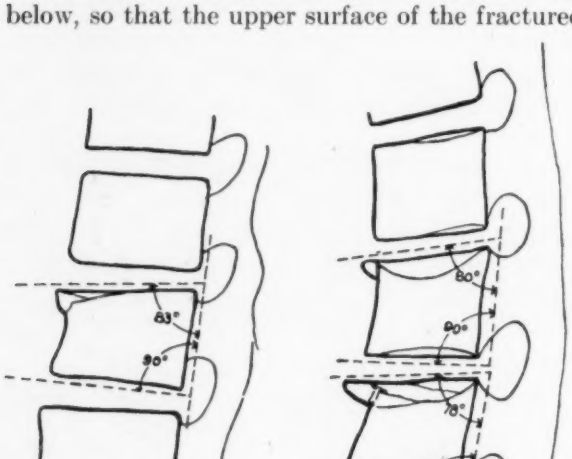


FIG. 2

Diagrams representing Potter's conception of the mechanics, resolution of forces, and deformity produced in varying grades of compression fracture. Note the preservation of integrity of the 90-degree angle between the posterior altitude and inferior and superior surfaces of the injured centra. (*Reproduced by permission of the U. S. Department of Labor from "The Wedge-Shaped Vertebra; Some Distinctions between Healed Fracture and Healed Vertebral Disease" by H. E. Potter. Bulletin No. 602, Bureau of Labor Statistics, 1934.*)

Considering the compressive force of each nucleus upon the terminal cartilage plate of the subjacent vertebra, one must remember that this is expansile since, as Petter has found in the fresh cadaver, the normal disc is confined under an average positive pressure of 30.2 pounds. Actually, combined with this compressive force, there is a rolling forward of the nucleus of each intervertebral space as the spine flexes forward and the vertebra moves slightly forward on the one beneath. This causes the exertion of the compressive force to be expended toward the anterior part of the upper surface of each vertebral body at the terminal ring, which is firmly anchored to the annulus.

One further reason that the lower half of each vertebral body is spared in a compression is suggested by Lange as due to greater cupping or depression of the under surface of each lumbar vertebra as compared to the upper surface which is almost flat. This increases with age, and also increases with each segment from above downward in the lumbar spine. The cupping spares the expenditure of force upon the anterior half of the lower-end surface of the vertebral body.

The normal cartilage plate is surprisingly strong and may not be disrupted, even though a trauma sufficient to produce a fracture of the adjacent vertebral bodies is exerted upon it.[12, 2]

In the last few years, much discussion has appeared relative to the physiology and pathology of the intervertebral disc in connection with spine injuries. Not so much attention has been given to the bony marginal ring or "*Randleiste*" which encircles the end plate. This ring is thicker in front, and is progressively larger as one travels down the spine, so that, in the anterior upper edge of a lumbar vertebra in the adult, it is a raised edge fused with the spongiosa of the vertebra and not more than 1.5 to 2 millimeters in height. The ratio between the anterior altitude of a lumbar vertebra and this marginal

ridge is about one to ten, and the vertebra will have attained the greatest height by the fifteenth year. These rings, even in the anterior part where they are widest, figure only 4 to 8 millimeters in width.

Hollis Potter has recently reviewed 500 consecutive compression fractures in which the arches were intact, and in only one case did he find a compression of the lower half of the vertebral body. The integrity of the lower half of the body is easy to recognize. Even in almost total destruction of the vertebral body, it will be noticed that in the upper half there is more condensation and more wedging deformity than in the lower half (Fig. 2).

In summary, a fractured vertebral body with arch intact does not simulate any other type of compression. If the posterior surface of the vertebral body in the lateral roentgenogram is taken as a base line, the upper surface will be found to form with it an angle more acute than a right angle, and the lower surface either retains its 90-degree relation with the base line, or is less affected than the upper half. Compression of the upper half is greater in the anterior than in the posterior altitude. If the fracture is not great, the posterior altitude is intact. The anteroposterior measurement of the upper surface is greater than that of the lower surface. Commonly, there is a displacement of the upper anterior corner, involving the terminal ridge and underlying spongiosa. In the anteroposterior view of recent fractures, the loss of anterior altitude is more difficult to recognize.

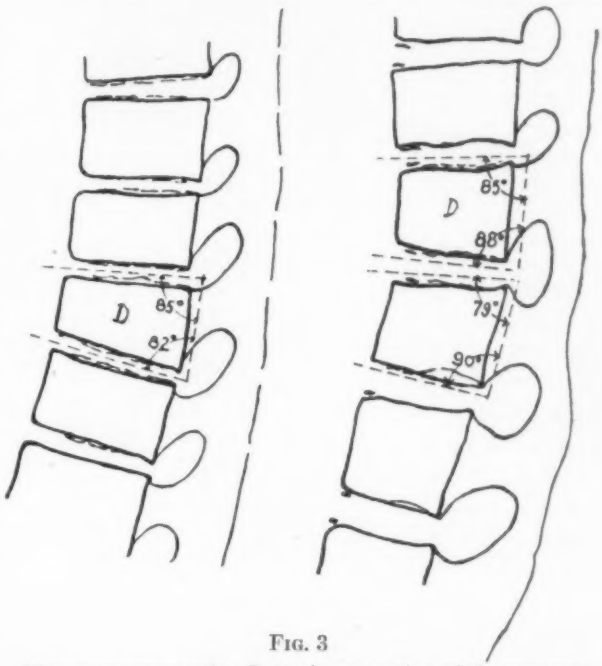


FIG. 3

Diagrams representing Potter's conception of the compression of centra in "osteochondritis". As soon as the distortion of the body is established, the terminal plates at both ends of the centrum are affected so that a compression altering the 90-degree angle relation of both terminal plates to the posterior altitude results. (*Reproduced by permission of the U. S. Department of Labor from "The Wedge-Shaped Vertebra; Some Distinctions between Healed Fracture and Healed Vertebral Disease" by H. E. Potter. Bulletin No. 602, U. S. Bureau of Labor Statistics, 1934.*)

In distinguishing between old healed compression fractures and healed non-traumatic lesions of vertebrae, it is found that, in a fracture, the wedged deformity is roughly preserved and still affects the upper half only, or the upper half out of proportion to the lower half of the body. The increased density in the upper half of the spongiosa may persist for years. The internal structure of the compressed bone seldom resumes the appearance of the unaffected lower half.

The width of adjacent intervertebral spaces is often unaffected in minor compressions.

In the extensively crushed vertebra, the irregular callus presents none of the characteristics of marginal osteophytes of spondylosis, extending as it does into the common ligament, and originating or becoming attached down the surface of the vertebral body far from the groove of the epiphysis where the osteophytes of spondylosis originate.

The research of Gaugele, Haumann, and Hellner, the observations of Lange, and the collection of Schmorl seem definitely to prove that general spondylosis and osteophyte production in the spine do not result from compression fracture of a vertebra.

Wedge-shaped deformities of the vertebrae, developing independent of trauma, may result from changes peculiar to adolescence. As roentgenographic technique improved, Scheuermann, in 1920 and in the years following, was able to trace the progress of the

pathology of the end plate and marginal ring through the steps of irregular calcification and failure of calcification, with increased density of the upper and lower portions of the vertebral body, to the final deformity and irregular calcification of the cartilaginous ring. Sometimes there is a persistence of a separate anterior portion of this ring, which has been called "Scheuermann's node", and this is easily mistaken for fracture. This node may occur with or without dorsal kyphosis.

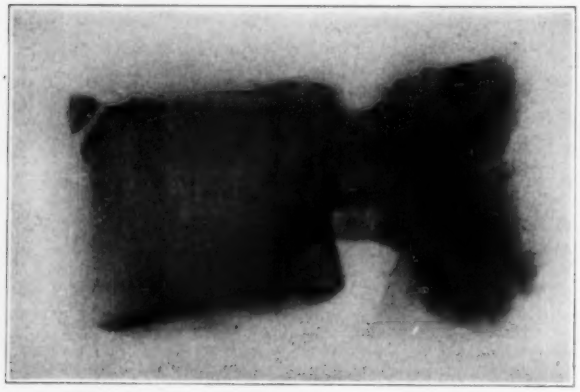


FIG. 4

Roentgenogram of a dissected vertebra, presenting a "Scheuermann's node".

The union of the terminal ring with the cartilaginous end plate and with the body of the vertebra should be complete between the twenty-second and twenty-fourth years.

The etiology of the condition is, of course, unknown. Schmorl and Junghanns found some tendency either to herniation into the vertebral bodies or to development of Schmorl's node in about 38 per cent. of 10,000 spines examined pathologically. Their followers have associated Scheuermann's disease and the subsequent wedging of vertebrae, with the result of prolapse of the disc into the body, diminishing the intervertebral space, and damaging the anterior portion of the growing centrum, where the increased pressure load is concentrated. Thus, the wedging of the bodies appears to them secondary to improper mechanical function of the degenerating and collapsing disc, following escape of the nucleus. However, no demonstrable collapse of the intervertebral space or disc herniation is routinely seen in roentgenographic studies of the disease.

The wedged body of juvenile osteochondritis presents the following characteristics distinguishing it from fracture:

1. The location of frequency is generally from the eighth to the tenth thoracic vertebrae, although occasionally a single vertebra, either thoracic or lumbar, may be involved.[13, 16] If a lumbar vertebra is affected, it is generally the fourth or the fifth.
2. The zone of change involves the upper and lower marginal rings and the end plate of the affected vertebra, and invariably in slighter degree those of adjacent vertebrae.
3. The general contour of the centrum is affected by compression of the ends, absence of cupping, and a wavy appearance due to irregular calcification of the ring.
4. Scheuermann's nodes and irregular beads of ring calcification may appear.
5. Subsequent kyphosis is not as angulated as in compression fracture, and it involves both ends of the centrum (Fig. 2).

Certain defects in development may be mistaken for fracture. In the first year of life, the vertebral body, in the lateral roentgenogram, appears as a rectangle with blunted corners; both the anterior and posterior borders of the terminal plates present a notching.

The significance of these steplike or notched spaces, and of later irregular ossification of this body, is important. This space is ringlike in form, extending entirely around the vertebral margin. The stepped space is lower toward the front than at the side or back. It is normally filled with cartilage, termed by Schmorl the "*Randleiste*", or it may be referred to as the "marginal ring".

In contrast to the adult vertebra, the ends of juvenile vertebral bodies show deep grooves extending radially toward the center of the end plates and cutting in more deeply

in front, those on the sides being reduced in length and depth. This change, described as "crenation"[25], may persist in adult life and be mistaken for the radial fracture lines of slight compression fracture.

The development of the marginal ring begins in small foci of calcification, as has been described by Schmorl and Junghanns, which appear in the female between the sixth and eighth years, and in the male between the seventh and ninth years. These first foci of calcification consolidate gradually into a single strip or band at about the twelfth year. In the fifteenth year, the ring becomes consolidated with the vertebral body. In the marginal ring are incorporated the "fibers of Sharpey", which attach it firmly to the annulus fibrosus. The vertebral body is completely developed by the twenty-fifth year. The marginal ledge is now entirely ossified and grown into the vertebral body. There is no cartilaginous plate between this ring and the spongiosa of the body.

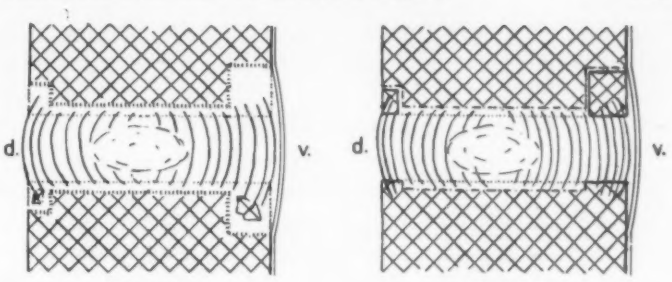


FIG. 5-A FIG. 5-B

Diagrams taken from Schmorl and Junghanns, representing sagittal sections through an intervertebral disc and adjacent vertebral bodies.

Fig. 5-A: The marginal ring, or *Randleiste*, of the inferior margin of the upper centrum is represented as a square blank space indicating a cartilaginous stage of development. This is also indicated as present in the cartilaginous end plate.

The outermost fibers of the annulus fibrosus are represented by curved lines penetrating into the *Randleiste*. These "Sharpey's fibers" extend across the intervertebral space to be embedded in the *Randleiste* of the vertebra below, which presents a stage of beginning ossification. There is no metaphyseal-growth region.

Fig. 5-B: The diagram represents a later stage of development. The *Randleisten* are completely replaced by bone in which the fibers of the annulus are firmly incorporated. The *Randleiste* has not yet fused with the body of the upper vertebra, as it has with the body of the lower vertebra. This lower vertebra presents the completely developed adult vertebra (after twenty-five years of age). No zone of cartilage persists between the "marginal ring" and the centrum. In section, these rings are at all stages of development more wedge-shaped than square, as they are represented in these diagrams. (*Reproduced from G. Schmorl und H. Junghanns: Die gesunde und kranke Wirbelsäule im Röntgenbild. Leipzig, Georg Thieme, 1932.*)

Schmorl and all recent observers consider that the marginal ring is purely functional, and that there is no true metaphyseal or epiphyseal bone growth in a vertebral body.

The persistence of a roughly triangular shadow in the lateral roentgenogram, seen in the region of the anterior upper corner of the vertebra and mistaken for fracture, was first described by Hanson in 1926, in connection with Scheuermann's adolescent kyphosis, as being of non-traumatic origin, and indicating a disease or disturbance of development rather than the result of trauma (Fig. 3). Before this important contribution, these changes, persisting from childhood, were generally called "marginal avulsions". In 1927, Mau discussed these changes, grouping them with fractures. As late as 1930, Herbert Junghanns raised the question of the possibility of a persistence of the so-called vertebral epiphysis. Before Junghanns' paper, and after Hanson's work appeared, Michajlow and Tscherepnina exhaustively discussed the persistence of a triangular rim at the lower anterior border of the fifth cervical vertebra, and Janker described seven cases occurring in the lumbar spine.

Roux and Gebhardt believe that the firm attachments of the capsule of the disc to the marginal ring may be explained by an avulsion fracture dating from childhood. Schajowicz suggested a damage of the blood supply of the marginal ring, due to an avulsion in childhood.

Since this defect of development (a separate triangular shadow in the lateral roentgenogram) appears most frequently in the upper anterior border of a lumbar vertebra, particularly the lower lumbar vertebrae, Junghanns and others believe that the stimulating cause might be traction exerted on the surface of the lumbar vertebrae by the pull of the natural lumbar lordosis, and might be due to abnormal exertion during the adolescent

period. To explain the growth of the Scheuermann's node resulting in a rim much thicker than the normal marginal ring, Delahaye quotes the law of Delpech: "Excessive pressure causes arrest or diminution of activity of epiphyseal cartilages and growth of bone and diminished pressure causes hyperactivity".

As late as 1920, these persisting islands were being reported in the German medical literature as sequelae of tuberculous spondylitis.[15] Hetzar's work in 1939 clarifies the distinction between the persistent rests of the terminal ring and true fractures.

Boerema, in a paper published in 1931, describes insufficiency of turgor of the nucleus as allowing compression of the annulus anteriorly and either subsequent deterioration of the anterior upper part of the vertebra and ring, or hypertrophy of the ring edge hardly distinguishable from spondylotic spurring.

Lyon describes a disease process in the marginal rings which he calls "osseous epiphyseal necrosis", comparing it to such diseases of the long bones as have been described by Perthes, Kienböck, Köhler, and Schlatter. He supposes that subchondral destruction of the metaphysis occurs; but since growth of a vertebra is not in any way comparable to the growth of a long bone, and since the terminal rings are in no way comparable to epiphyses, there is no reason for such an analogy here. In development, the terminal rings do not correspond to the epiphyses in the time or manner of their formation. There is no type of growth in the vertebral body.[26]

The following are the criteria of differential diagnosis of these Scheuermann's nodes from vertebral fractures:

1. The localization is important. The nodes are found most frequently in the lower lumbar region, particularly in the upper border of the fifth vertebra. Compression fractures are commonest in the upper lumbar and lower thoracic vertebrae.
2. The zone of separation is often wide and clear and, like the node, has smooth edges; whereas, irregular edges are seen in the compression fracture.
3. The node is often larger in size than a marginal ring fragment would be.
4. The dislocation of the fragment or node is often considerable; it may be somewhat upward and anterior. Dislocation of a marginal fracture is slight and generally downward and anterior, corresponding to the plane of compression of the superior surface of the body of the vertebrae. The general contour of the centrum or vertebral body remains normal in a case of a persistent node; but, in a case of fracture, it varies from slight compression of the upper surface to a wedge-shaped deformity.
5. The mechanogenesis of the persistent node may be abnormal lumbar-extension strain or distraction in childhood, and not a single injury; whereas, a compression fracture is the result of flexion of the spine, a shearing force, and an impression of force on the disc and the upper surface of the centrum, leaving its typical mark.

REFERENCES

1. Boerema, I.: Über Kyphosis dorsalis adolescentium. Arch. f. Klin. Chir., CLXVI, 737, 1931.
2. Compere, E. L., and Keyes, D. C.: Roentgenological Studies of the Intervertebral Disc. A Discussion of the Embryology, Anatomy, Physiology, Clinical and Experimental Pathology. Am. J. Roentgenol., XXIX, 774, 1933.
3. Delahaye, André: Etude sur quelques Diagnostics du Mal de Pott, en particulier sur l'Epiphysite Vertébrale de Croissance. Thèse de Paris, Legrand, 1924.
4. Gaugele: Wirbelkallus und Spondylosis deformans. Verhandl. d. Deutschen Orthop. Gesellsch., XXVI, 247, 1931.
5. Hanson, Robert: Some Anomalies, Deformities and Diseased Conditions of the Vertebrae during Their Different Stages of Development, Elucidated by Anatomical and Radiological Findings. Acta Chir. Scandinavica, LX, 309, 1926.
6. Hanson, Robert: Einige Röntgenstudien über "Normal" Rücken während der Wachstumsjahre. Fortschr. a. d. Geb. d. Röntgenstrahlen, XXXIX, 1079, 1929.

7. HAUMANN, W.: Die Wirbelbrüche und ihre Endergebnisse. Stuttgart, Ferdinand Enke, 1930.
8. HELLNER, HANS: Wirbelfrakturen und Spondylitis deformans. Arch. f. Orthop. u. Unfall-Chir., XXIX, 417, 1931.
9. HETZAR, W.: Die traumatischen Schädigungen der bandscheibennahen Wirbelkörperabschnitte. Deutsche Ztschr. f. Chir., CCLII, 396, 1939.
10. JANKER, ROBERT: Die Epiphysen der Wirbelkörper und ihre Veränderungen. "Persistierende Wirbelkörperepiphysen". Fortschr. a.d. Geb. d. Röntgenstrahlen, XLI, 597, 1930.
11. JUNGHANNS, HERBERT: Gibt es "persistierende Wirbelkörperepiphysen"? Fortschr. a. d. Geb. d. Röntgenstrahlen, XLII, 704, 1930.
12. KEYES, D. C., AND COMPERE, E. L.: The Normal and Pathological Physiology of the Nucleus Pulposus of the Intervertebral Disc. An Anatomical, Clinical and Experimental Study. J. Bone and Joint Surg., XIV, 897, Oct. 1932.
13. KLEINBERG, SAMUEL: Lumbar Vertebral Epiphysitis. Arch. Surg., XXX, 991, 1935.
14. LANGE, MAX: Die Wirbelgelenke. Die röntgenologische Darstellbarkeit ihrer Krankhaften Veränderungen und ihre Beziehungen zu den Verschiedenen Erkrankungen der Wirbelsäule. Stuttgart, Ferdinand Enke, 1934.
15. LEHRNBECHER, A.: Über seitliche Wirbelaufnahme bei Spondylitis tuberculosa. Fortschr. a. d. Geb. d. Röntgenstrahlen, XXVII, 643, 1920.
16. LINDEMANN, K.: Bandschiebenverknöcherung bei juvenilen Kyphosen. Verhandl. d. Deutschen Orthop. Gesellsch., 30 Kongress, 1935 (Beilageheft, Ztschr. f. Orthop. LXIV, 143). Stuttgart, Ferdinand Enke, 1936.
17. LYON, ERNST: Über Brüche von Epiphysen der Lendenwirbelkörper im Jünglingsalter. Fortschr. a. d. Geb. d. Röntgenstrahlen, XXXVIII, 376, 1928.
18. MAU, C.: Die Kyphosis dorsalis adolescentium im Rahmen der Epiphysen- und Epiphysenlinienerkrankungen des Wachstumsalters. Ztschr. f. Orthop. Chir., XLVI, 145, 1924.
19. MICHAJLOW, M., UND TSCHEREPNINA, M.: Über die Wirbelknorpelinseln und deren Röntgenbild. Fortschr. a. d. Geb. d. Röntgenstrahlen, XL, 1061, 1929.
20. NIEDNER, FRANZ: Zur Kenntnis der normalen und pathologischen Anatomie der Wirbelkörperrandleiste. Forstschr. a. d. Geb. d. Röntgenstrahlen, XLVI, 628, 1932.
21. PETTER, C. K.: Methods of Measuring the Pressure of the Intervertebral Disc. J. Bone and Joint Surg., XV, 365, Apr. 1933.
22. POTTER, H. E.: (1) The Wedge-Shaped Vertebra; (2) Some Distinctions between Healed Fracture and Healed Vertebral Disease. Bulletin, U. S. Bureau of Labor Statistics, No. 602, 96, 1934.
23. ROUX AND GEBHARDT: *Quoted by* FRANZ NIEDNER.
24. SCHAJOWICZ, F.: Contributo alla struttura microscopica e alla patologia dei dischi intervertebrali nei giovani. Chir. d. Org. di Movimento, XXIV, 5, 1938.
25. SCHEUERMANN, H.: Kyfosis dorsalis juvenilis. Ugesk. f. Laeger, LXXXII, 385, 1920.
26. SCHMORL, G.: Zur Kenntnis der Wirbelkörperepiphyse und der an ihr vorkommenden Verletzungen. Arch. f. Klin. Chir., CLIII, 35, 1928.
27. SCHMORL, G., UND JUNGHANNS, H.: Die gesunde und kranke Wirbelsäule im Röntgenbild. Leipzig, Georg Thieme, 1932.

MARCH FRACTURE

Report of a Case Involving Both Fibulae

BY LIEUTENANT COLONEL A. SCOTT HAMILTON AND MAJOR HOWARD E. FINKLESTEIN

Medical Corps, Army of the United States

From the Surgical Service, Station Hospital, Camp Claiborne, Louisiana

Interest in the study of march fractures in this country has been greatly stimulated by the training program of our Armed Forces. The incidence of these fractures has increased beyond any previously reported number. In the Station Hospital, Camp Claiborne, Louisiana, numerous cases involving the metatarsals have been observed, but this experience has not paralleled that of Ollonqvist and Hannson, both of whom have reported many cases involving the tibia and femur. The discovery of a case involving both fibulae therefore seemed to warrant more than casual consideration.

CASE REPORT

S. W. D., a private in the infantry, was inducted into the Army of the United States, December 2, 1942. After induction, the soldier participated in training of gradually increasing severity. On December 28, 1942, he had participated in a fifteen-mile hike, and on January 1, 1943, he reported on sick call, complaining of pain and swelling about the lateral aspect of both ankles. The right ankle caused the greater discomfort, and it alone showed a tendency toward inflammatory rubor of the skin. A diagnosis of "shoes too tight" was made, and hot soaks and liniment were employed as treatment.

The soldier again reported on sick call, January 6, 1943, and was referred to the Orthopaedic Clinic, Station Hospital, as a possible case of arthritis. No history of accidental injury was elicited. Symptoms were confined almost wholly to the right ankle. There was no palpable bony tumefaction. Roentgenographic examination of the part demonstrated a line of increased density (Fig. 1) which was interpreted as an incomplete fracture (question of Looser's "*Umbauzone*" [3,4]).

The patient was admitted to the Hospital on January 8, 1943.

Physical examination showed him to be of the asthenic type, seventy-two inches in height, and weigh-

Fig. 1

Roentgenogram of the right ankle taken January 6, 1943.

Fig. 2

Roentgenogram of the left ankle taken March 4, 1943.

ing 165 pounds. He had been a music director before induction and had led a purely sedentary life. The blood pressure was 128 systolic, and 76 diastolic. Urine and blood analyses were within normal limits. The temperature was 98.4. A moderate myopia was present. Other physical findings did not deviate from normal, except for the right lateral malleolus. The skin over this region was oedematous and slightly discolored. Tenderness was moderate, but no crepitus could be elicited. Motions of the ankle and subtalar joints were uninhibited. A walking cast was applied.

On January 16, 1943, the patient was discharged with the walking cast in place. This was removed in the Out-Patient Clinic on February 1, 1943. The recurrence of symptoms in the right ankle was noted two weeks later, after a six-mile march. The patient was given light duty but, in spite of this, the left ankle began to become troublesome by February 21, 1943.

The soldier reported on sick call, March 1, 1943, and was again referred to the Orthopaedic Clinic. Roentgenograms of both ankles were taken on March 4, 1943, and the following report was submitted: "Roentgenograms of the left ankle show callus formation about the fracture site at the distal end of the fibula. The callus is extensive and may be sufficient for the resumption of function. Could this be classified as a march fracture? This is almost identical with the place in which we saw a fracture without displacement in the right ankle on January 6, 1943." Roentgenographic re-examination on March 9, 1943, resulted in the following report: "Examination of both ankles shows considerable bony callus at the fractures of the distal ends of the fibulae. The fragments are in good position and alignment, and the fractures are fairly well stabilized."

By March 13, 1943, the right ankle was asymptomatic, but the left ankle was still moderately painful. Symptoms in the latter were aggravated by continued exercise. Whirlpool and massage gave moderate relief.

On May 15, 1943, the patient stated that he had been symptom-free for the past month. A slight fusiform swelling remained in the region of the lesion on each side.

Recently, the patient returned for further treatment, complaining of his right foot. Roentgenographic examination revealed a march fracture involving the second metatarsal.

COMMENT

While it is not our intention to draw any conclusions from a single case, we feel that the disability suffered by this soldier is identical with that of Case 3 described by Weaver and Francisco. Furthermore, we are of the opinion that in all probability it is also identical with the cases described by Ollonqvist, by Hannson, and by Peterson. We did not and, in retrospect, do not consider that chronic inflammation, as suggested by Roberts and Vogt, other than that of a non-specific nature, has any bearing on the etiology or pathogenesis. We agree with Camp and McCullough that this entity probably does not represent a new disease, but is simply the response of the weight-bearing skeletal frame to continuous overstrain.

REFERENCES

1. Camp, J. D., and McCullough, J. A. L.: Pseudofractures in Diseases Affecting the Skeletal System. Radiology, XXXVI, 651, 1941.
2. Hannson, C. J.: On Insufficiency Fractures of Femur and Tibia. Acta Radiol., XIX, 554, 1938.
3. Looser: Über pathologische Formen von Infraktionen und Callusbildungen bei Rachitis und Osteomalakie und anderen Knochenerkrankungen. Zentralbl. f. Chir., XLVII, 1470, 1920.
4. Looser, E.: Über Spätrachitis und Osteomalacie. Klinische, röntgenologische und pathologisch-anatomische Untersuchungen. Deutsche Ztschr. f. Chir., CLII, 210, 1920.
5. Ollonqvist, L. J.: Osteopathia Itineraria Tibiae. (*In* Proc. Northern Assn. Med. Radiol.) Acta Radiol., XVIII, 526, 1937.
6. Peterson, L. T.: March Fracture of the Femur. Report of a Case. J. Bone and Joint Surg., XXIV, 185, Jan. 1942.
7. Roberts, S. M., and Vogt, E. C.: Pseudofracture of the Tibia. J. Bone and Joint Surg., XXI, 891, Oct. 1939.
8. Weaver, J. B., and Francisco, C. B.: Pseudofractures. A Manifestation of Non-Suppurative Osteomyelitis. J. Bone and Joint Surg., XXII, 610, July 1940.

MARCH FRACTURES

BY CAPTAIN BAXTER L. CLEMENT

Medical Corps, Army of the United States

From the Station Hospital, Basic Training Center No. 7, Army Air Forces Training Command, Atlantic City, New Jersey

Since the etiology of march fractures has been a debatable issue for some time, a study has been made of a series of thirty-two cases*, all confirmed roentgenographically, which have been encountered in a Basic Training Center Station Hospital over a period of five months. In view of this relatively large number of cases, it seemed that some valuable data concerning the character of such fractures might be compiled. The individuals affected were undergoing their first few weeks of military training, and undoubtedly were being subjected to unusual foot strain in comparison with their civilian life. Their average length of military service was eleven weeks. The symptoms were typical, consisting of localized tenderness directly proximal to the metatarsophalangeal joint of the metatarsal involved, and swelling on the dorsum of the foot. Since fractures of this type result in a loss of time from duty of from six to ten weeks or even longer, an investigation of their possible etiology was undertaken for its intrinsic value, and also to determine the practicability of devising some mechanical means of reducing their incidence.

Anteroposterior and oblique roentgenograms of the feet were routinely taken. An oblique view was found to be necessary for the diagnosis of very early march fractures; they cannot be detected on a direct anteroposterior view. In extremely early cases, the fracture line was identifiable only as a very faint oblique nick extending proximally through the cortex of the metatarsal shaft. In no case was a transverse fracture line seen.

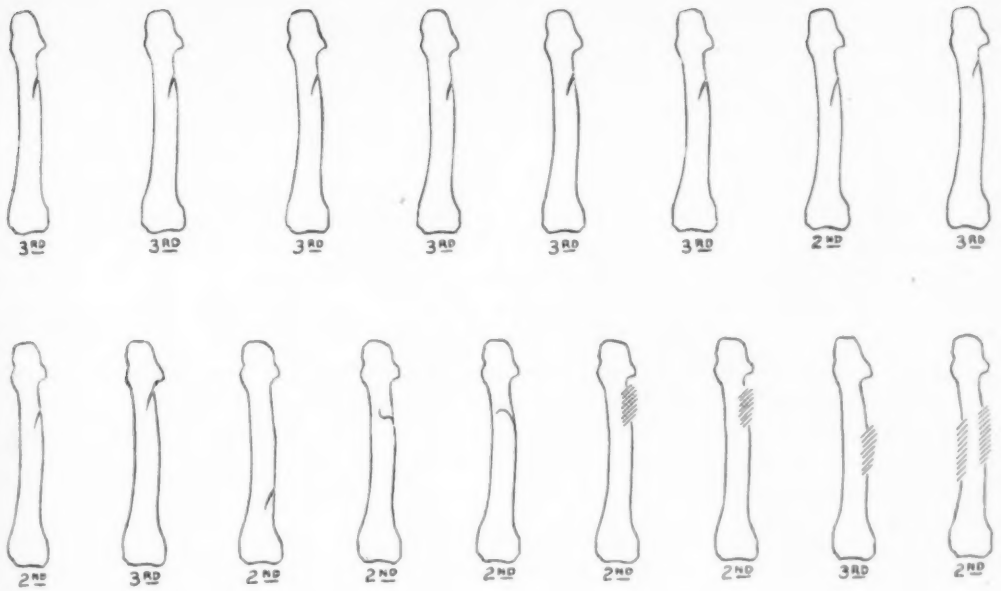
Figure 1 is a diagrammatic representation of the location of the fractures in the series studied. It will be noted that, in the thirty-two cases, the left foot was involved twenty times and the right foot twelve times. Only the second and third metatarsals were affected, the second in seventeen cases and the third in fifteen cases, showing an essentially equal distribution. Approximately 75 per cent. of the fractures were in the distal or the middle third of the metatarsal shaft. In all but three cases the fracture line appeared on the medial aspect of the shaft.

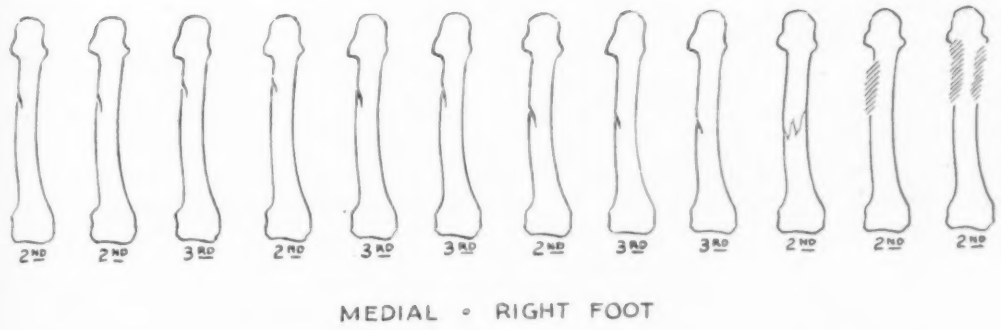
The average age of the patients was twenty-six years, the youngest eighteen years, and the oldest forty-five years. From our observations, the weight of the patient could not be considered a predisposing factor. The fracture occurred in an approximately equal proportion of persons with flat feet and in those with a normal arch.

Approximately 100 roentgenograms of normal feet were examined in order to note the location of the sesamoid bones, and the distance between the first and second metatarsals. However, these factors appeared to have no significance when the normal roentgenograms were compared with the proved cases of fracture.

There are many theories regarding the etiology of march fractures. Most of the evidence points to a physiologically weakened foot.[1] Normally, the weight of the body is borne chiefly on the medial surface of the foot and a tripod is formed by the heads of the first and fifth metatarsals and the os calcis[2], with the first metatarsal acting as a splint for the second and third metatarsals. However, in all but two of our cases, it was noted from the roentgenograms that the second and third metatarsals were longer than the others. This would destroy the normal tripod arrangement and structurally weaken these metatarsals. It is suggested that, in a foot in which the second and third metatarsals are longer

* This series of cases was collected from the Orthopaedic Service, of which Major John E. McDonald is Chief. Valuable assistance was given by Captain Norman Heilbrun, Chief of the Roentgenological Service.





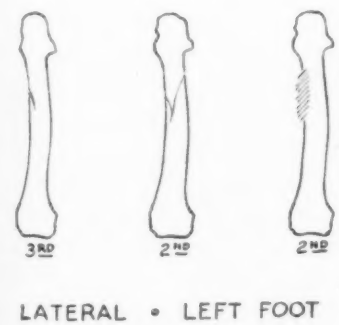


FIG. 1.

Diagrammatic representation of the location of fractures, seventeen being on the medial aspect of the left foot, twelve on the medial aspect of the right foot, and three on the lateral aspect of the left foot. In twenty-seven cases, which were recognized early, the obliquity of the fracture lines is clearly evident. In seven cases, which were not seen early, the fractures, represented by the heavily shaded areas, were probably originally oblique fractures.

than the others, march fractures may occur from stress and strain as a result of abnormal bony leverage and spasm of the irritated interossei dorsales and lumbricales muscles. That these factors play a rôle is suggested by the fact that the fracture line was oblique in all of our cases, and usually appeared on the dorsomedial aspect of the shaft.

It has been our practice when a march fracture is suspected clinically, even if the initial roentgenogram appears negative, to apply a metatarsal pad for the support of the second and third metatarsals, and to prescribe complete rest. This is especially desirable when the roentgenogram shows the second and third metatarsals to be longer than the first. In our experience, this procedure limits the period of total disability, should a fracture later be identified roentgenographically.

SUMMARY

Thirty-two cases of march fracture encountered in a Basic Training Center have been studied in an attempt to determine the etiology, the characteristics, and a possible mechanical means of reducing the incidence.

In view of the present generally accepted beliefs regarding march fractures, the following observations are of striking interest:

1. The fracture line was oblique in all early cases.
2. The second and third metatarsals only were involved.
3. The fracture line appeared on the medial surface of the metatarsal in twenty-nine of the thirty-two cases.
4. The second and third metatarsals were of greater length, as compared with the first metatarsal, in all but two cases.

In a clinically typical march fracture, which has not progressed sufficiently to be detected in a roentgenogram, the application of a metatarsal pad and the prescription of complete rest will shorten the total period of disability.

CONCLUSION

In our group of cases the second and third metatarsals were longer than the remaining metatarsals, thereby deranging the normal tripod structure of the bones of the foot. It therefore appears that march fractures occur from stress and strain, as a result of the leverage of the bones and the muscle pull of the lumbricales and interossei dorsales muscles, which become spastic from the irritation induced by marching.

REFERENCES

1. Meyerding, H. W., and Pollock, G. A.: March Foot. The Military Surgeon, LXXXVI 593, 1940.
2. Wilson, Philip D.: Fractures of the Bones of the Foot. *In* A Textbook of Surgery. By American Authors. Edited by Frederick Christopher. Ed. 3, p. 749. Philadelphia, W. B. Saunders Company, 1942.

DELAYED REDUCTION OF FRACTURES *

BY JOHN R. MOORE, M.D., PHILADELPHIA, PENNSYLVANIA

From the Temple University Hospital and Medical School, Philadelphia

INTRODUCTION

From August 1, 1934, until January 15, 1935, all fractures and dislocations at this Hospital were treated as emergencies. Patients with 253 fractures were admitted to the Accident Dispensary during this period. The author personally supervised the majority of the reductions, regardless of the time of day or night that the patient appeared. Numerous difficulties confronted the literal interpretation and application of immediate or emergency reduction. Delay of one sort or another frequently occurred. Among the chief causes were shock, head injury, swelling, anaesthetic danger, and, last but not least, personnel inadequacy (orthopaedic, roentgenographic, anaesthetic, or nursing). The general plan for handling fractures and dislocations at this period was similar to the current method, except that emergency reduction was substituted for the present delayed reduction. Tables have been prepared for the analyses of cases treated during the early period, but, because of the similarity to those found in delayed reduction, they have been purposely omitted to save time and space, and for emphasis.

The general policy of immediate reduction was discontinued February 15, 1935. It was recognized as impractical, possibly unnecessary, and led to needless expenditure of energy and equipment.

The time for reduction of fractures has been a moot question throughout the ages. Hippocrates [8] (born 460 B.C.) and his contemporaries favored immediate reduction, but recommended delay (seven to twenty-one days) if swelling and inflammation were present. Orbasius (born 325 A.D.) advocated delayed reduction (third or fourth day or thereafter). The pendulum swung back and forth until the renaissance of Hippocratism during the sixteenth, seventeenth, and eighteenth centuries. Immediate reduction found more or less universal application during this period. In the latter part of the eighteenth century, the question again arose regarding reduction in the presence of swelling. Boyer (born 1757) and Larrey (born 1766) were champions of immediate reduction, but emphasized vigorously the contra-indications,—swelling and muscle spasm. Malgaigne (born 1806) leaned distinctly toward delayed reduction. "It is known that . . . inflammation always of itself delays the reparative process; . . . the organization of the callus does not begin until the second [stage of consolidation]. One may, therefore, . . . allow the first period to elapse before proceeding to reduction." Toward the latter part of the nineteenth century the pendulum swung back again to immediate reduction. Practically all textbooks and teachings urged its universal adoption. Dur-

FIG. 1

Routine examination for circulation and sensation prior to immediate immobilization.

* Read at the Annual Meeting of The American Orthopaedic Association, Cleveland, Ohio, June 7, 1943.

FIG. 2

Reduction of Pott's fracture on the seventh day after fracture. Note the absence of swelling. A cylindrical section of the cast has been removed and the plaster edges have been turned back. Following reduction, the plaster defect was repaired easily and quickly by plaster reinforcement. Multiplane fluoroscope (E.W.C.) was substituted for x-ray.

ing the last World War, Bancroft attributed the marked improvement in fracture results to the immediate traction applied on the battlefield and early reduction upon arrival at the hospital. Since that period, up to and including the present time, fractures have been regarded as emergencies more or less universally. Immediate reduction with bone contact, adequate immobilization, and early mobility of joints and soft parts, are the primary fundamentals of the orthodox-treatment concept.

DELAYED REDUCTION

Beginning February 15, 1935, the following plan for the handling of fractures has been in use in the Temple University Hospital and Medical School of Philadelphia:

FIG. 3-A

Through cylindrical windows, single half-pins are inserted into the upper and lower fragments of a fractured tibia and fibula.

FIG. 3-B

The pins have been incorporated in the plaster, and a cylindrical section has been removed at the site of the fracture. Notice the absence of swelling. The reduction was done on the seventh day.

FIG. 3-C

The reduction has been completed and the defect in the plaster has been closed.

1. All fractures are examined immediately for nerve and vessel injury, and for such complications as head, spinal-cord, and internal injuries, *et cetera*. The treatment of these major problems is instituted immediately by the department concerned.

2. All fractures of the long bones are immobilized immediately in plaster-of-Paris splints which routinely include the joint above and the joint below.

3. Roentgenograms are taken at the convenience of the Roentgenographic Department, either immediately or the following day.

4. One day a week is set aside for the reduction of fractures (Tuesday), hence the local sobriquet, "Tuesday fractures".

Following immobilization at the time of admission, the patient is kept under observation in the Accident Dispensary for an hour, and is then allowed to return home, or is admitted to the Hospital, depending upon the absence or presence of complications, facilities for care at home, *et cetera*. He is instructed to report on the day set aside for reduction, and special emphasis is placed upon pre-anaesthesia preparations. On the morning of the reduction, a cylindrical section of plaster, three to four inches in width, is removed

TABLE 1

DISTRIBUTION OF FRACTURES

Location	No. of Cases
Tarsus, metatarsus, and phalanges	574
Ankle (malleoli, anterior and posterior lip of tibia)	585
Tibia and fibula (shaft)	631
Knee (tibial tuberosities, femoral condyles, tibial spines, and tibial tubercles)	187
Femur	449
Hip (trochanter, neck and head of the femur, and acetabulum)	129
Pelvis	121
Spine (body, transverse process, and lamina)	474
Ribs	384
Hand (carpals, metacarpals, and phalanges)	555
Wrist (lower end of radius and ulna)	882
Radius and ulna (shaft)	764
Elbow (humeral condyles, olecranon, and head of the radius)	310
Humerus	445
Shoulder (clavicle, scapula, and head of the humerus)	519
Total	7,009

at the site of the fracture in fractures of the long bones. If traction is required to maintain reduction, a single pin is inserted in each major fragment. The temporary splints employed in other types of fractures are removed. These latter fractures are reduced and are subsequently splinted according to the standard methods. Reduction is uniformly successful, provided complete muscle relaxation is obtained. Generally speaking, the effort required for reduction is greater than that observed in immediate reduction, yet a closed reduction is obtainable in the majority of cases; open reductions are comparatively few (Table II). Following the reduction, the patient is permitted to go home or is admitted to the Hospital depending primarily upon the status prior to reduction.

CONTRA-INDICATIONS TO DELAYED REDUCTION

1. Compound fractures are regarded as emergencies. The suppression of hemorrhage, immediate immobilization in plaster, supportive treatment for shock, the adminis-

FIG. 4-A

An immobilized fracture of the femur. Circular windows are being cut for insertion of the half-pins.

tration of prophylactic sera, chemotherapy, *et cetera*, form the usual routine. Immediate débridement and reduction follow, except in the cases of patients with critical complications,—such as head injury, internal injury, and severe shock. Incidentally, forty-five compound fractures were necessarily delayed, aside from immobilization and other routine treatment, and were reduced along with the simple fractures without any evidence of

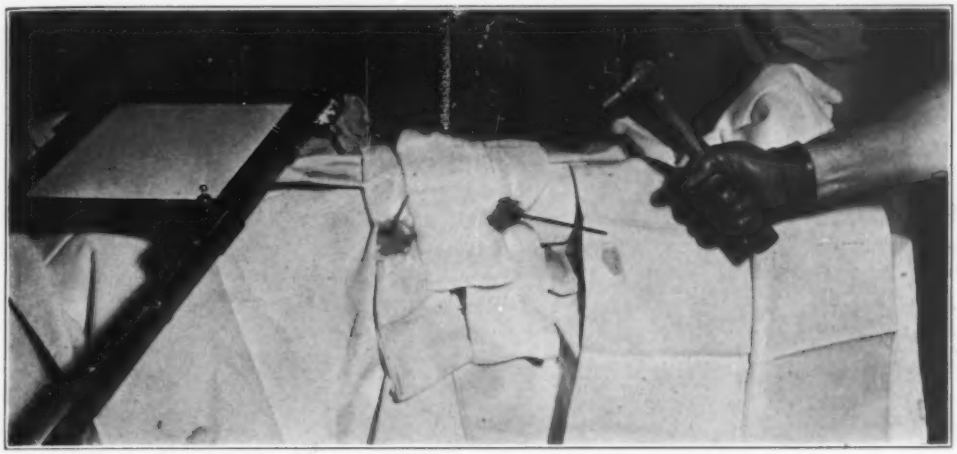


FIG. 4-B

The pins, which have been inserted, were drilled through the proximal cortex, and gently drilled or tapped into the distal cortex.

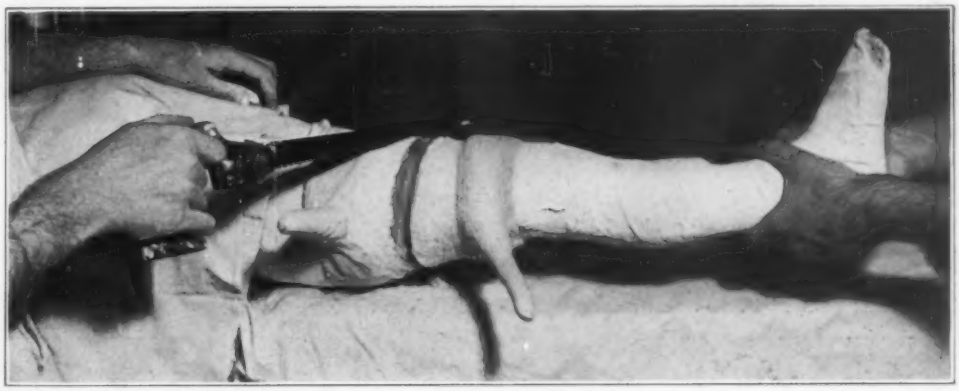


FIG. 4-C

The pins have been incorporated in plaster, and the cylindrical section has been cut out; the reduction is in process.

FIG. 4-D

The fracture has been reduced and the defect in the plaster has been closed. The pins have been incorporated in the cast.

deleterious effects. The author does not advocate delayed reduction of compound fractures, but wishes to point out that delayed reduction has been necessarily employed in this group without any noticeable ill effects regarding repair, infection, union, and function.

2. Fractures with major nerve or vessel injury are immobilized immediately, preferably in plaster, and are reduced immediately. Immediate attention to the complication is directed by the department concerned.

3. Simple dislocations (not compound or complicated by fracture) are regarded as emergencies. Fracture-dislocations at the ankle, elbow, shoulder, and other joints have been uniformly delayed.

CASES STUDIED

This study is based upon 7,009 fractures which have been admitted to the Accident Dispensary of Temple University Hospital between August 1, 1934, and May 1, 1943.

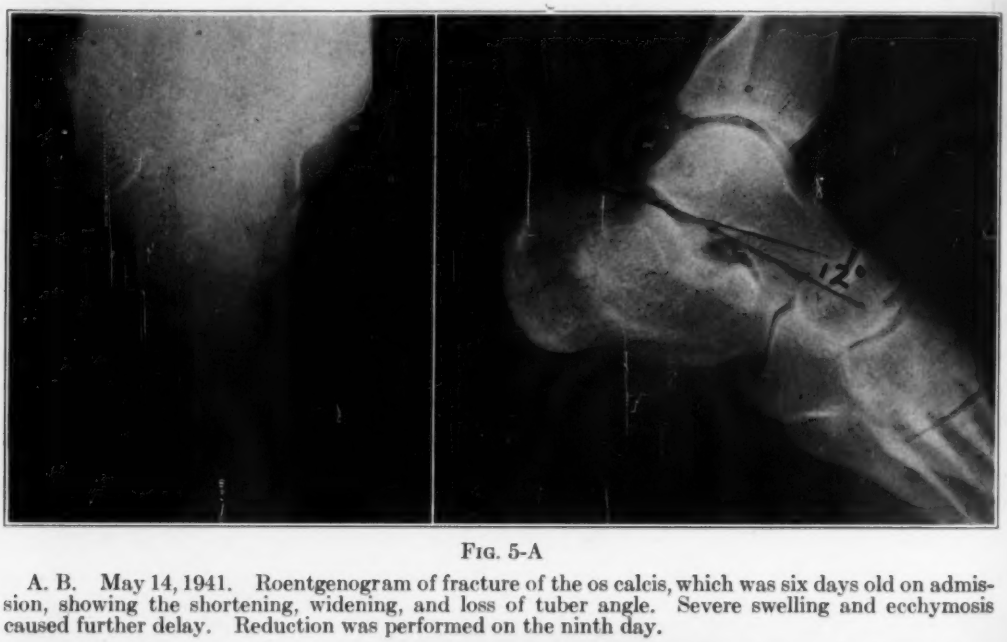


FIG. 5-A

A. B. May 14, 1941. Roentgenogram of fracture of the os calcis, which was six days old on admission, showing the shortening, widening, and loss of tuber angle. Severe swelling and ecchymosis caused further delay. Reduction was performed on the ninth day.

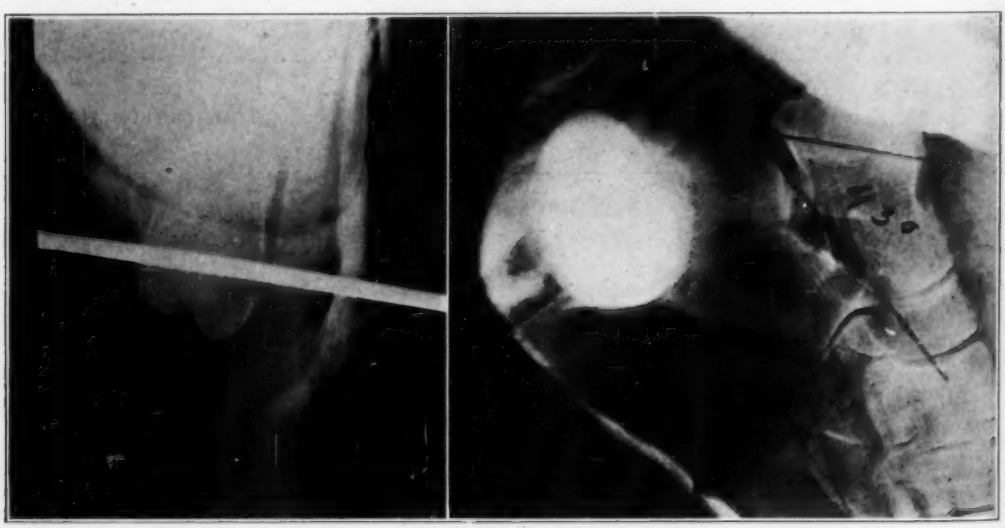


FIG. 5-B

May 17, 1941. Fracture of the os calcis after reduction.

FIG. 5-C

July 15, 1941. Fracture of the os calcis eight and one-half weeks after reduction.

Reduction was required in 5,608 fractures. The value of immediate immobilization cannot be too strongly stressed. Fourteen hundred and one fractures in the present series required no reduction, merely immobilization. The author feels that the immediate immobilization upon admission partially accounts for the lack of displacement, and, therefore, the lack of need of reduction in this large percentage of cases. The distribution of the fractures is shown in Table I.

Roentgenographic Examination

Roentgenograms are taken immediately after the fracture is immobilized, or, provided there are no unusual complications, at the convenience of the Roentgenographic

FIG. 6-A

B. P. Fracture and dislocation of the talus. Reduction was performed on the ninth day.

FIG. 6-B

Fracture of the talus one year after reduction. Note the complete restoration and normal contour.

Department, the time varying from immediately after the immobilization to the following day.

Anaesthesia

A general anaesthetic is employed in the majority of cases. Nitrous oxide and oxygen, supplemented by avertin and pentothal sodium are considered most efficacious.

Type of Reduction

Of the 5,248 simple fractures requiring reduction, fifty-six were reduced by open methods and 5,189 by closed.

Time of Reduction

Immediate reductions of simple fractures numbered 253, and of compound fractures, 315. Delayed reductions of simple fractures numbered 4,936, and of compound fractures, forty-five. All of the delayed reductions were done from four to eleven days after fracture.

FIG. 7-A

E. H. December 15, 1937. Bimalleolar fracture before reduction. Reduction was performed on the seventh day.

FIG. 7-B

April 16, 1938. Bimalleolar fracture four months after reduction. Normal function has been restored.

Ease of Reduction

More effort was required to reduce fractures which were delayed than those which were reduced immediately. Fractures reduced between the fourth and the eleventh day were distinctly easier to handle than those reduced in twenty-four, thirty-six, or forty-eight hours. Reduction was obtainable in the delayed cases (fourth to eleventh day) with comparatively few open procedures. The fractures which required open reduction are shown in Table II. It should be noted that the vast majority of open procedures recorded are those generally conceded to be advisable because of the character of the fractures.

The author's experience does not confirm the observations made by Hein. "The percentage of perfect reductions drops very rapidly until the 24 to 48 hour

FIG. 8-A

J. K. Anteroposterior and lateral views of a fracture of the middle third of the tibia. The reduction was performed on the eighth day after fracture.

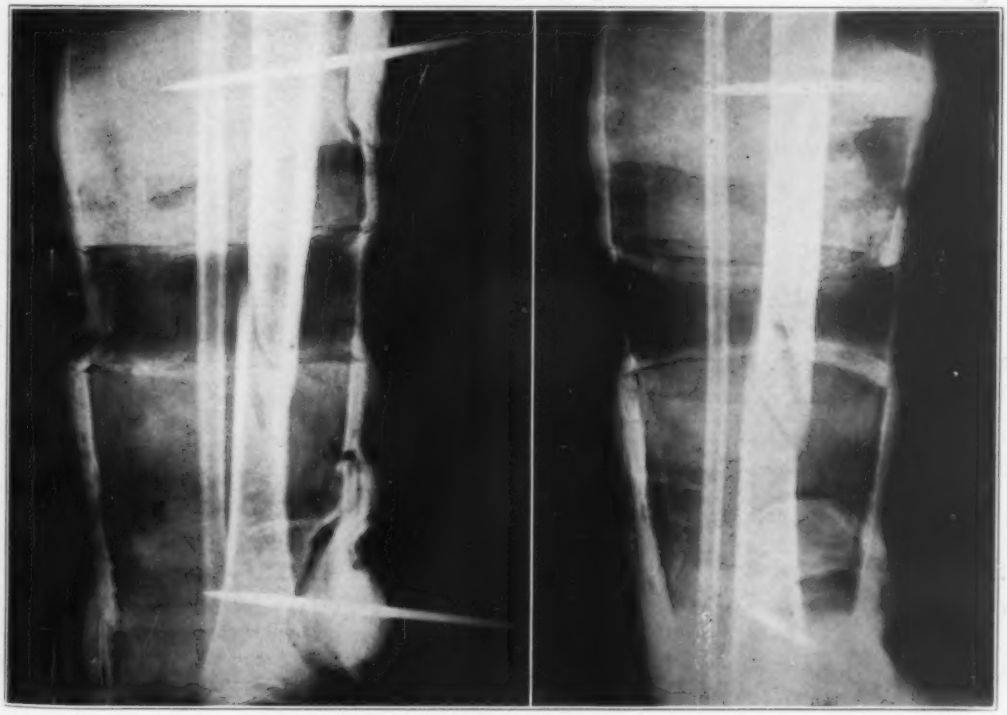


FIG. 8-B

The fracture has been reduced. Note the pin fixation.

FIG. 8-C

The fourth month after reduction.

group only showed 4 per cent. . . . The increasing difficulty with reductions, together with increasing degrees of displacement following reduction as the time elapse increases is caused by: the congealment of the hemorrhage, . . . increasing edema of the soft parts . . . and muscle spasm." It is highly probable that Hein did not resort to immediate, complete immobilization during this delayed-reduction regimen.

The present author made the following observations concerning swelling:

Early swelling—up to four hours—was due usually to hemorrhage at the site of the fracture. Delayed swelling—four hours to ten days or longer—was due to the following:

1. Hemorrhage at the site of fracture,
2. Reaction of the tissues at the site of fracture to complications secondary to trauma,
3. Swelling of the part distal to the fracture, due to obstruction of venous and lymphatic return.

These changes were originally observed during the era of the fracture box. Immediate and total immobilization of the fracture, including the joint above and the joint below, at the time of admission reduced all of these factors markedly. Hemorrhage apparently subsided in a few hours. (This observation was based on the lack of pain.) Secondary swelling reached its peak in forty-eight hours, and subsided sufficiently to permit reduction in ninety-six hours. Appreciable swelling at the site of the fracture was rarely observed between the fourth and eleventh days. Figures 5-A and following illustrate the fractures which were reduced in from four to eleven days.

TABLE II

DISTRIBUTION OF FIFTY-SIX SIMPLE FRACTURES REQUIRING OPEN REDUCTION

Location		No. of Cases
Femur		2
Patella		10
Tibia and fibula		5
Metatarsals		2
Humerus,—	greater tuberosity	1
	surgical neck (head dislocated)	2
	supracondylar	4
	internal epicondyle	4
	external epicondyle	3
Forearm,—	radius (shaft)	6
	radius (head)	6
	ulna (shaft)	2
	olecranon	8
Metacarpals		1
Total		56

FIG. 9-A

FIG. 9-B

Fig. 9-A: M. McE. November 15, 1937. Fracture of the external tuberosity of the tibia and fracture of the neck of the fibula.

Fig. 9-B: November 19, 1937. Anteroposterior view immediately after reduction which was performed on the fourth day.

FIG. 9-C

November 19, 1937. Lateral view immediately after reduction.

FIG. 9-D

December 28, 1937. Six weeks after reduction.

Period of Repair

Tables III and IV illustrate the average time of immobilization, and the time required for union and the restoration of function. The incidence of malunions and non-unions was not remarkable. The analysis of the results obtained in the period in which immediate reductions were done (August 1, 1934, to February 15, 1935) gave identical results. The author questions the accuracy of this analysis because the period of immobilization was the same for both groups. It is possible that had the fractures been examined at various weeks after the reduction, a difference in the time required for repair might have been noted. However, since immobilization was of the same duration, no differences in the time required for repair were observed.

FIG. 10-A

M. R. September 11, 1938. Supracondylar fracture of the femur.

FIG. 10-B

March 24, 1939. The seventh month after reduction. Reduction was done on the seventh day.

FIG. 11-A

FIG. 11-B

Fig. 11-A: K. K. November 12, 1938. Fracture of the middle third of the femur.
Fig. 11-B: November 23, 1938. Anteroposterior view after reduction which was performed on the tenth day.

FIG. 11-C

FIG. 11-D

Fig. 11-C: November 23, 1938. Lateral view after reduction.
Fig. 11-D: May 22, 1939. Six months after reduction.

FIG. 12-B

December 7, 1938. Six months after reduction.

FIG. 12-A

H. D. June 6, 1938. Fracture of the middle third of the femur in Paget's disease.

FIG. 13-A

C. S. Pathological fracture of the upper third of the femur in Ewing's tumor. Reduction was performed on the tenth day.

FIG. 13-B

The third month after reduction.

Complications

In the study of the complications it is especially striking, and should be emphasized, that there were no instances of nerve complications—either sensory or motor—Sudeck's atrophy, or Volkmann's ischaemic paralysis following immobilization or reduction. Three of the latter were recognized on admission, and vascular surgery was instituted. There were two instances of embolism. One, which occurred during the third week after

FIG. 14-A

H. T. Paratrochanteric fracture of the femur. Reduction was performed on the fifth day.

FIG. 14-B

The fourth month after reduction.

reduction of a Pott's fracture, terminated fatally. A second patient recovered from three pulmonary infarcts during the sixth week after reduction of fractures of the shafts of the tibia and fibula. The two cases of embolism appeared so long after the time of fracture, that it seems only fair to regard the occurrence as a coincidence rather than as attributable to the fracture management. Three cases of occlusion of the brachial artery were observed but were easily recognized prior to plaster immobilization. In spite of the fact that no attempt was made to reduce fractures at the time of admission (immobilization in *status quo*) no secondary compounding was observed. However, it must be noted that no fractures were immobilized with the skin under tension, the alignment being corrected sufficiently to eliminate skin necrosis (secondary compounding).

FIG. 15-A

FIG. 15-B

A. B. October 1, 1940. An intracapsular fracture of the neck of the femur. Reduction was performed on the sixth day.

FIG. 15-C

FIG. 15-D

February 11, 1941. The fifth month after reduction.

Delayed union occurred in eight cases of tibial fractures (union not complete four months after fracture). Three of these cases had complete union after an additional immobilization period of two months. Five were not united at the end of six months and required sliding bone grafts. In seventy-eight cases of fracture of the neck of the femur,

FIG. 16-A

J. R. June 14, 1936. Capital fracture of the femur and comminuted fracture of the pubic rami. Reduction was done on the tenth day.

FIG. 16-B

October 30, 1937. Seventeenth month after reduction.

FIG. 17-A

A. S. Central dislocation of the head of the femur and fracture of the ilium. Reduction was performed on the thirteenth day.

FIG. 17-B

Twelfth month after reduction.

union occurred in sixty-seven and non-union in eleven. Studies were based on two-year follow-up. Three of the successful unions went on to aseptic necrosis in the head. Union was delayed until eight months in four fractures of the shaft of the femur.

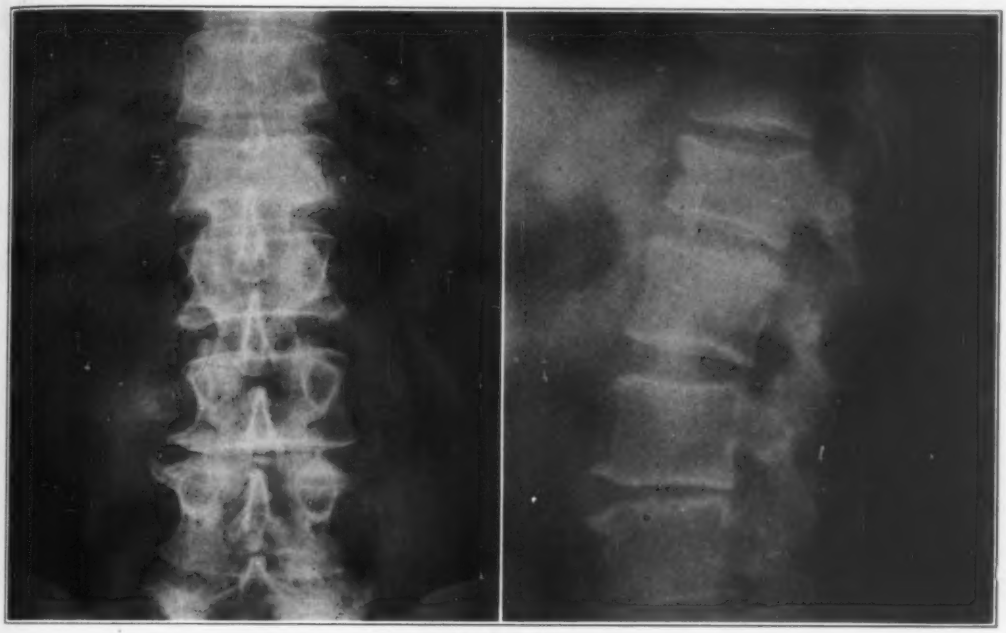


FIG. 18-A

G. Y. September 16, 1937. Compression fracture of the first lumbar vertebra. Reduction was done on the fourth day.

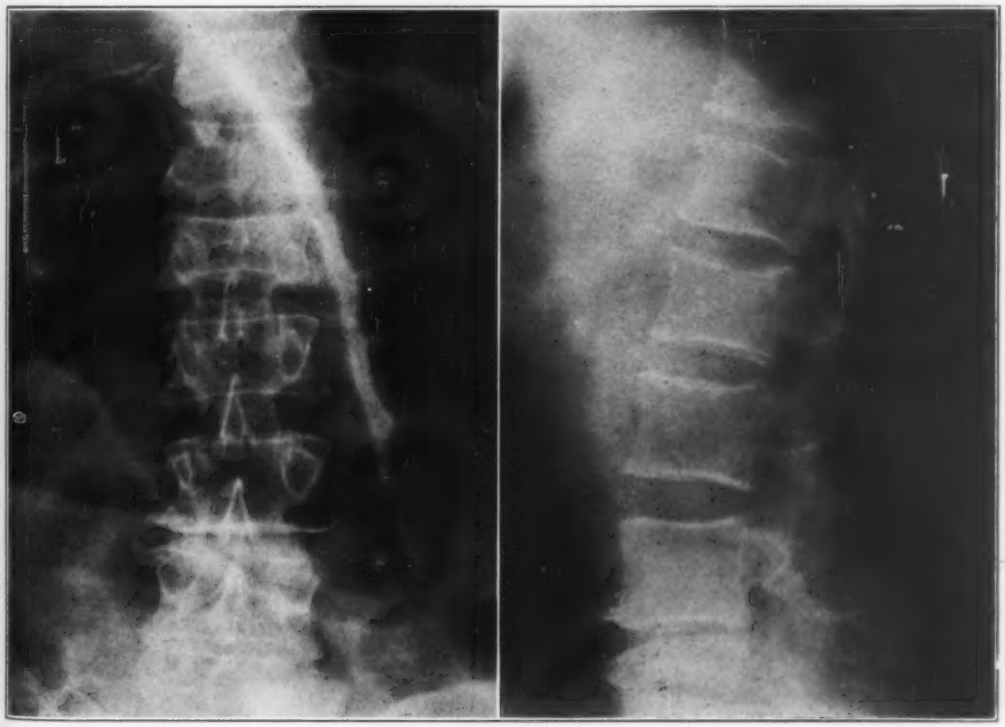


FIG. 18-B

May 20, 1938. The ninth month after reduction,

FIG. 19-A

T. G. January 3, 1938. Slipped lower radial epiphysis. Reduction was performed on the fifth day.

FIG. 19-B

February 8, 1938. One month after reduction.

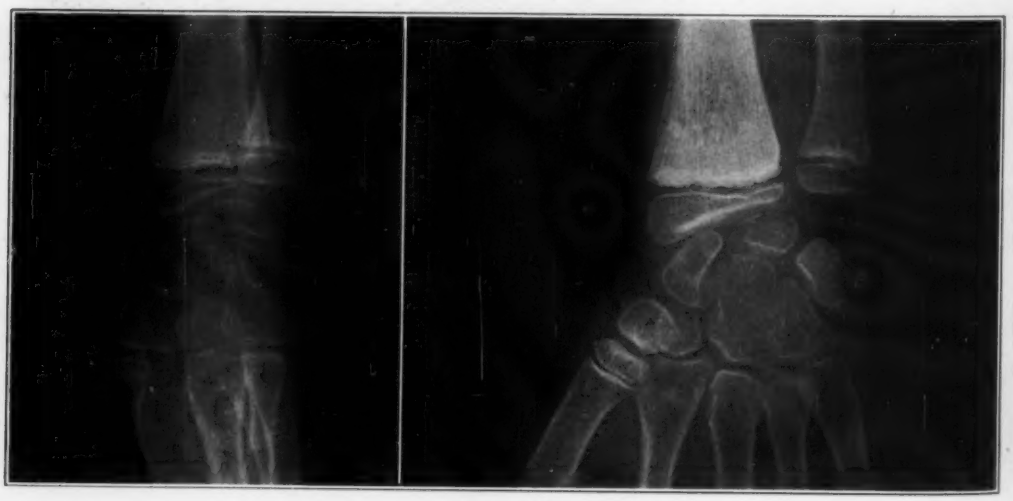


FIG. 19-C

The eighteenth month after reduction.

There were no cases of malunion sufficiently severe to require correction. Mild coxa vara and shortening were accepted in intertrochanteric fractures. Occasional mild lengthening occurred in fractures of the femoral shaft in children. One case of cubitus varus followed the removal of the fragment in a fracture of the internal condyle of the humerus.

One might naturally wonder how the patient would feel toward delayed reduction. Complaints have been few indeed. At no time have they been sufficient to require immediate reduction. Pain at the site of the fracture ceases almost at once following immobilization. It offers no more of a problem, as a rule, than the pain observed after

FIG. 20-A

A. W. September 14, 1937. Fracture of the lower end of the radius (Colles'). Reduction was done on the sixth day.

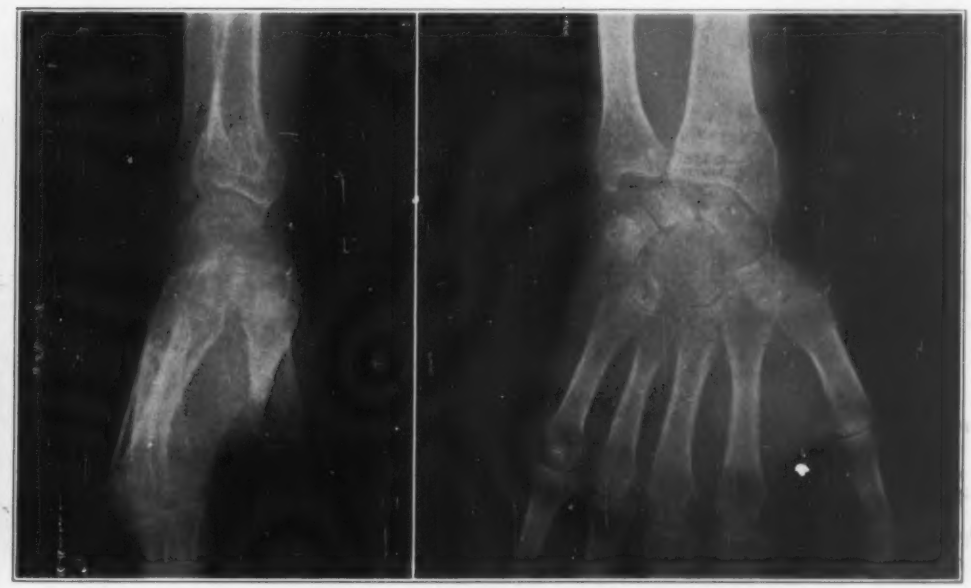


FIG. 20-B

January 8, 1938. The fourth month after reduction.

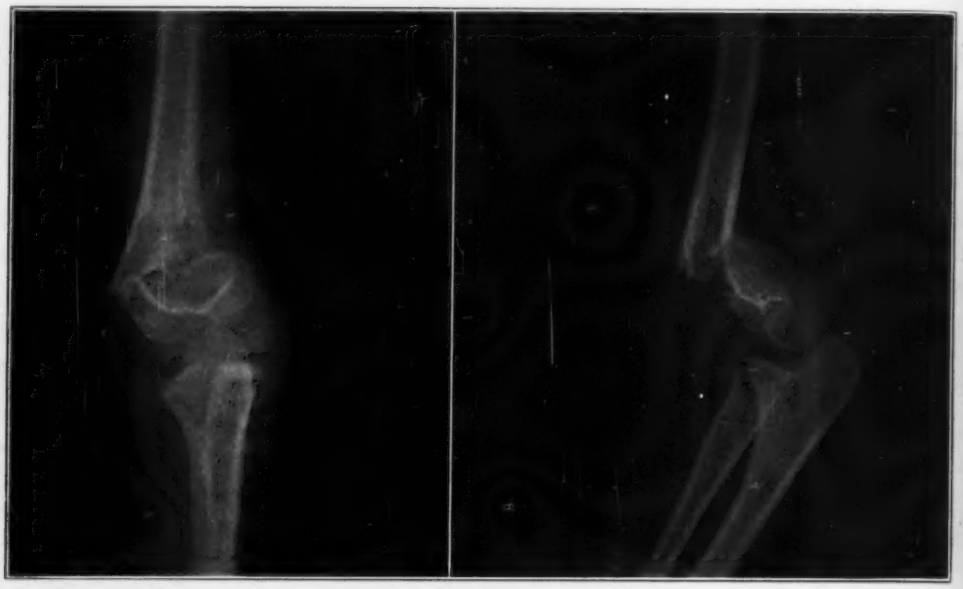


FIG. 21-A

E. R. October 15, 1939. Supracondylar fracture of the humerus. Reduction was done on the seventh day.

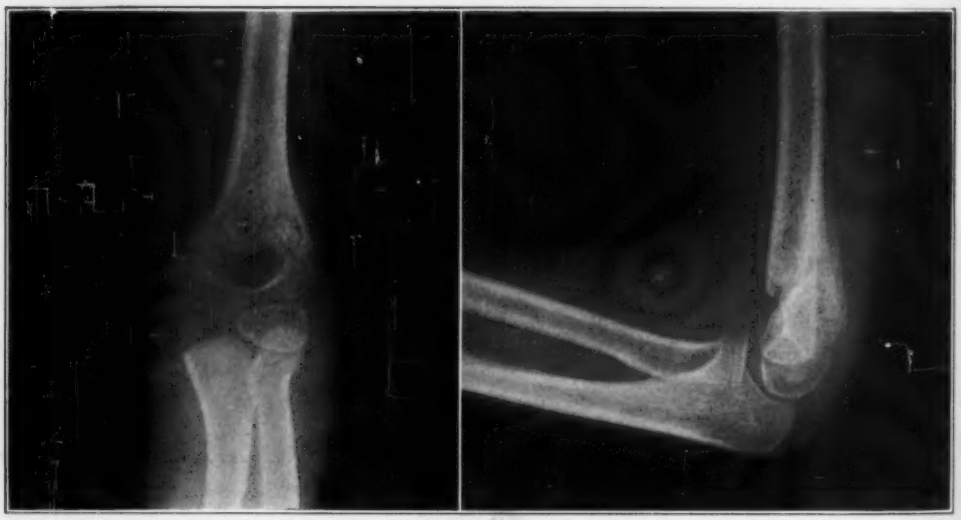


FIG. 21-B

Fourth month after reduction.

FIG. 21-C

February 15, 1940. The fifth month after reduction, showing flexion.

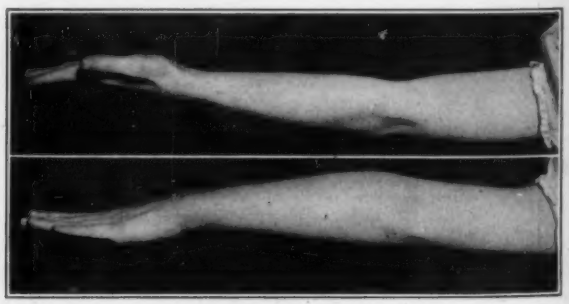


FIG. 21-D

February 15, 1940. The fifth month after reduction, showing range of extension.

reduction of the fracture. In general, patients return for reduction on the day set, without excitement, and readily cooperate in every respect.

FIG. 22-A

V. V. Fracture of the lower third of the humerus. Reduction was done on the seventh day.

FIG. 22-B

The fifth month after reduction.

TABLE III

AVERAGE TIME REQUIRED FOR REPAIR OF FRACTURES IN ADULTS

Location of Fracture	Immobilization	Union	Restoration of Function
Phalanges	4 weeks	6 weeks	Third month
Metacarpals	6 weeks	6 to 8 weeks	Third month
Radius and ulna	12 weeks	12 weeks	Fifth month
Elbow	3 weeks	8 weeks	Third to fifth month
Humerus,—shaft	12 to 16 weeks	12 to 16 weeks	Fifth month
neck	3 weeks	8 weeks	Fourth month
Clavicle	6 weeks	8 to 10 weeks	Twelfth week
Scapula	4 weeks	6 to 8 weeks	Fourth month
Spine	6 months	4 months	Sixth month
Metatarsals	6 weeks	8 weeks	Eighth to tenth week
Tarsals and os calcis	5 weeks	12 weeks	Sixth month
Ankle,—Pott's, trimalleolar, and bimalleolar fractures	5 weeks	12 weeks	Fourth to sixth month
Tibia and fibula,—shaft	12 to 16 weeks	12 to 16 weeks	Fifth to sixth month
Knee,— tuberosities and condyles	5 weeks	12 to 16 weeks	Sixth to ninth month
patella	2 weeks	8 to 12 weeks	Fourth to sixth month
Femur,—shaft	16 to 20 weeks	16 to 20 weeks	Sixth to seventh month
trochanter	12 to 16 weeks	16 to 20 weeks	Sixth to eighth month
neck	(Smith-Peter-sen nail)	6 months	Sixth to twelfth month
Pelvis	12 to 16 weeks	16 to 20 weeks	Sixth month

TABLE IV

AVERAGE TIME REQUIRED FOR REPAIR OF FRACTURES IN CHILDREN

Location of Fracture	Immobilization	Union	Restoration of Function
Phalanges	3 weeks	5 weeks	Eighth to tenth week
Metacarpals	4 weeks	5 to 7 weeks	Eighth week
Radius and ulna	8 weeks	8 weeks	Third month
Elbow	3 weeks	6 weeks	Third to fourth month
Humerus,—shaft	8 to 12 weeks	8 weeks	Third month
neck	8 weeks	8 weeks	Twelfth week
Clavicle	4 weeks	6 weeks	Sixth week
Scapula	3 weeks	5 weeks	Fifth week
Metatarsals	4 weeks	6 weeks	Sixth to eighth week
Tibia and fibula,—shaft	8 weeks	8 weeks	Tenth to twelfth week
Femur,—slipped upper epiphysis	4 months	6 months	Sixth to ninth month
slipped lower epiphysis	8 weeks	10 weeks	Fourth month
shaft	8 to 12 weeks	10 weeks	Twelfth to sixteenth week
base of neck	12 weeks	12 weeks	Fourth to sixth month
Pelvis	6 weeks	8 weeks	Third to fourth month

CONCLUSIONS

The reduction of fractures may be delayed. The ideal time for delayed reduction is between the fourth and eleventh day. Complete immobilization in plaster should precede delayed reduction. Compound fractures (exceptions noted), simple dislocations, compound dislocations, and fractures complicated by nerve or vessel injury, should be regarded as emergencies. The time required for repair in the group of delayed reductions would seem to date from the time of fracture, rather than from the time of reduction, since additional delay was not observed. Union and function appear to be unhampered. Delayed reduction provides the opportunity for excellent teamwork. The best-trained

individuals are readily available for all purposes. The patient is adequately prepared for anaesthesia. Medical students have the opportunity to observe fractures before, during, and after reduction. The author has not had the opportunity of running a parallel series over the eight-year period, but, comparing the results of this delayed-reduction group with the previous, admittedly small, immediate-reduction group, there seems to be little doubt as to the preponderance of merit. Delayed reduction is not urged here. If teams which provide well-trained supervision are available for immediate reduction, immediate reduction should be done. In the localities where well-trained supervision is not at hand, it would appear that delayed reduction is by far the better procedure. In conclusion, one might say in defense of delayed reduction, "a well-supervised delayed reduction is better than a poorly supervised immediate reduction".

The author is indebted to W. Edward Chamberlain, M.D., Professor of Radiology of the Temple University Medical School, for his helpful suggestions and for his constructive criticism.

REFERENCES

1. Bancroft, F. W.: The General Question of the Emergency Treatment of Fractures. Ann. Surg., C, 843, 1934.
2. Boyer, Alexis: *Quoted by* Malgaigne.
3. Bromfeild, William: Chirurgical Observations and Cases, Vol. 2, p. 58. London, T. Cadell, 1773.
4. Crocq, J.: Du traitement des fractures des membres. Mémoire couronné. Bruxelles, J. B. de Mortier, 1851.
5. Hein, B. J.: The Time Element in the Reduction of Fractures. Ohio State Med. J., XXXIV, 1339, 1938.
6. Heinemann, Bernhard: Ein historischer Überlick über die Therapie der Knochenbrüche. (Inaugural Dissertation.) Bonn, Anton Brand, 1937.
7. Larrey, D. J.: *Quoted by* Malgaigne.
8. Malgaigne, J. F.: A Treatise on Fractures. (Translated from the French by J. H. Packard with notes and additions) p. 167. Philadelphia, J. B. Lippincott, 1859.
9. Orbasius: *Quoted by* Malgaigne.

DISCUSSION

Dr. M. N. Smith-Petersen, Boston, Massachusetts: We have heard Dr. Moore talk about this many times. In good hands this is a method that works out extremely well and satisfactorily. It is a dangerous procedure in untrained hands.

Dr. George E. Bennett, Baltimore, Maryland: I would prefer to have an expert reduce a fracture ten days afterward than an amateur the first day.

Dr. Kellogg Speed, Chicago, Illinois: I have heard Dr. Moore give a talk along this line before a little different audience. Everyone appreciated his magnificent results. My impression this morning was that many of his results were shown in children where we invariably expect to get good results. I agree with Dr. Smith-Petersen that it would be very unwise to teach this method or advocate it in general for practitioners. When you come to analyze the average run of fractures over the country, how many are reduced immediately? I venture to say it is a very small percentage. A great many of them lie six, eight, or ten days, either for consultation purposes or because there is no help available, and yet the results are good. I believe Dr. Moore put it rather strongly, to come out so bluntly and say we must delay. I agree with Dr. Bennett that it is far better to have a good man reduce a fracture after a few days than to have a poor man do it immediately.

Dr. E. W. Ryerson, Chicago, Illinois: I belong to the older school which has opposed for many years the use of pin fixation, and yet in the last two years I have seen so many cases reduced and held firmly that perhaps I must begin to change my opinion about it. The only comment I wish to make is that if you are going to put one pin in the lower fragment and one pin in the upper fragment, why not do a complete job and put two pins in each fragment and then you will have an accurate and firm fixation.

Dr. J. Albert Key, St. Louis, Missouri: I have always felt that this method was one of expediency rather than of choice. I cannot believe that a patient is as comfortable in a cast or without a cast, with a fracture not reduced, as the same patient would be with the same degree of fixation if the fracture were reduced. If this is not a method of expediency, it seems to me there should be an optimum time,—that is, the fracture should not be reduced on Tuesday but at the optimum time.

Dr. Moore's results shown here are beautiful; they are better than mine. On the other hand, I think they are a lot better than I could get with his method. In my opinion, fractures that have gone for several days with a lot of swelling are more difficult to reduce than if reduction had been done immediately. Part

of Dr. Moore's success is due to a principle, which all of us realize but rarely use, that if you immobilize each fragment in a cast and then cut the cast, reduction is easier. This is especially true if you want to put in a couple of pins and then fix those in the cast. Personally I am going to stick to the method which I have always used,—that is, I prefer to see the patient as soon as possible after the accident, and reduce the fracture immediately.

DR. H. W. ORR, LINCOLN, NEBRASKA: I believe that a fracture should be reduced immediately to relieve the complication of muscle spasm. I am for immediate reduction at all times.

DR. WALTER P. BLOUNT, MILWAUKEE, WISCONSIN: I have no criticism of the technique which expediency has dictated, but I should not like to let this subject go unchallenged in its application to fractures in children. We know that a fracture of the femur in a child has an average overgrowth of one-half inch in the first year. If the fracture is accurately reduced as to length, the femur is likely to be half an inch too long. If two pins are inserted, the overgrowth is going to be increased. I think the use of these pins in children is not to be encouraged. I would like very much to know the end results of Dr. Moore's cases in children, to find out the extent of overgrowth in every case, not in a few months but in two years.

DR. RYERSON: Is that overgrowth permanent? I think it disappears.

DR. BLOUNT: I have a paper ready for publication in which I present the end results of cases followed for ten years or until the epiphyses are closed. The overgrowth is permanent. I have done leg-shortening operations on femora with too much length when the fracture was originally perfectly reduced. It is not uncommon to see an overgrowth of an inch in a fracture with one-half inch of initial overriding. The growth stimulation from pins may be an undesirable sequel. Skeletal fixation is rarely needed in a child, and it introduces an added risk.

DR. J. R. MOORE, PHILADELPHIA, PENNSYLVANIA (closing): I wish to thank all the discussors. I agree with Dr. Smith-Petersen that delayed reduction is a dangerous problem unless the principles of fracture treatment, bone contact, proper immobilization, and adequate blood supply, are adhered to strictly.

Dr. Speed apparently is under the impression that this paper has been presented for the purpose of insisting upon delayed reductions. On the contrary, the primary purpose of this paper is to establish the fact that delayed reductions can be done, and that healing and recovery apparently progress as in the cases of immediate reduction.

Dr. Ryerson has asked why not employ two pins in each fracture fragment. We have not done so simply because we have found one pin per fragment combined with plaster sufficient. In fractures of long bones in infancy and in early childhood, the pin-and-plaster method is not employed if one of the more conservative conventional methods will suffice. In late childhood and adolescence, the pin-and-plaster procedure has been used routinely without significant complications of any type.

Dr. Key has asked whether or not delayed reductions were purely a matter of expediency. Immediate reduction was practiced at Temple University Hospital for six months and delayed reduction has been practiced for eight years. The principal reasons for continuing delayed reduction of fractures are emphasized in the concluding section of this paper.

Dr. Blount has asked the question concerning overgrowth due to the stimulating effect of the pins. Overgrowth has been an uncommon occurrence.

BRACHIALGIA. A MANIFESTATION OF VARIOUS LESIONS

BY I. WILLIAM NACHLAS, M.D., BALTIMORE, MARYLAND

GENERAL CONSIDERATIONS

Brachialgia is a symptom complex characterized by pain in and about the shoulder with radiations down the arm. It is essentially a sensory disturbance, and has been described under various names that have usually been based on the etiological concepts presented by various investigators. Brachialgia means pain in the arm, and has no reference to the causative factor.

This syndrome is produced by an irritation of the sensory nerve tracts somewhere between the cortical sensory cells and the areas in which the pain is felt. From the points of nerve irritation the sensation is projected peripherally as radiations,—that is, it is interpreted by our cerebrum as coming from the distal ends of the nerves. A diagram of the nerve tracts supplying the sensation to the shoulder and arm is found in Figure 1. Theoretically it is possible to produce these symptoms by irritating the nerves (1) within the skull, (2) where the skull joins the spine, (3) within the cervical spinal column, (4) in the intervertebral foramina, (5) in the supraclavicular region, (6) in the shoulder, and (7) in the thorax. Actual clinical experience has shown the symptom complex to be associated with (a) thalamic tumors, (b) platybasia, (c) tumors of the cervical spinal cord, (d) herniations of the intervertebral disc, (e) lesions within the intervertebral foramina, (f) cervical rib, (g) aneurysms of the subclavian artery, (h) pressure in the supraclavicular region, (i) injuries about the shoulder, (j) superior sulcus tumors, and (k) heart lesions.

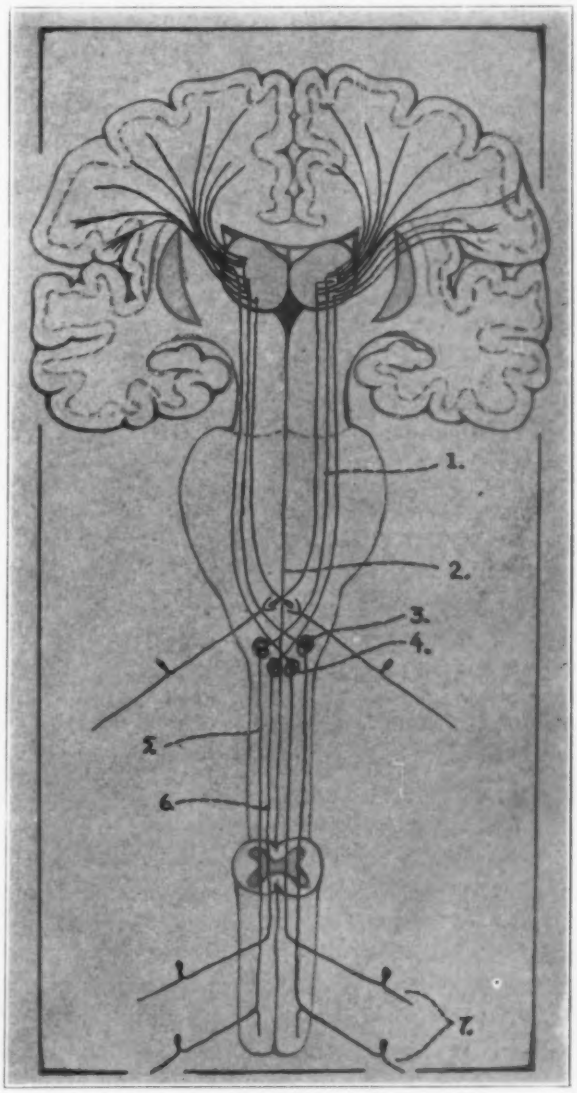


FIG. 1

The sensory tract: (*1*) medial lemniscus; (*2*) sensory decussation; (*3*) nucleus cuneatus; (*4*) nucleus gracilis; (*5*) fasciculus cuneatus; (*6*) fasciculus gracilis; and (*7*) posterior nerve roots. (*Modified after Poirier in Gray's Anatomy of the Human Body. Edited by Warren H. Lewis. Ed. 24, p. 869, Fig. 773, Philadelphia, 1942, and reproduced through the courtesy of Lea & Febiger.*)

Within the skull and within the spinal column the tracts are well protected by a good bony casing, so that only severe trauma can act as a disturbing factor from without. On the other hand, neoplasms, inflammatory lesions, and degenerative conditions can affect this tract without external factors. At the junction between the skull and the spine, and at the joints between the various vertebrae, the protective casing is not rigid,

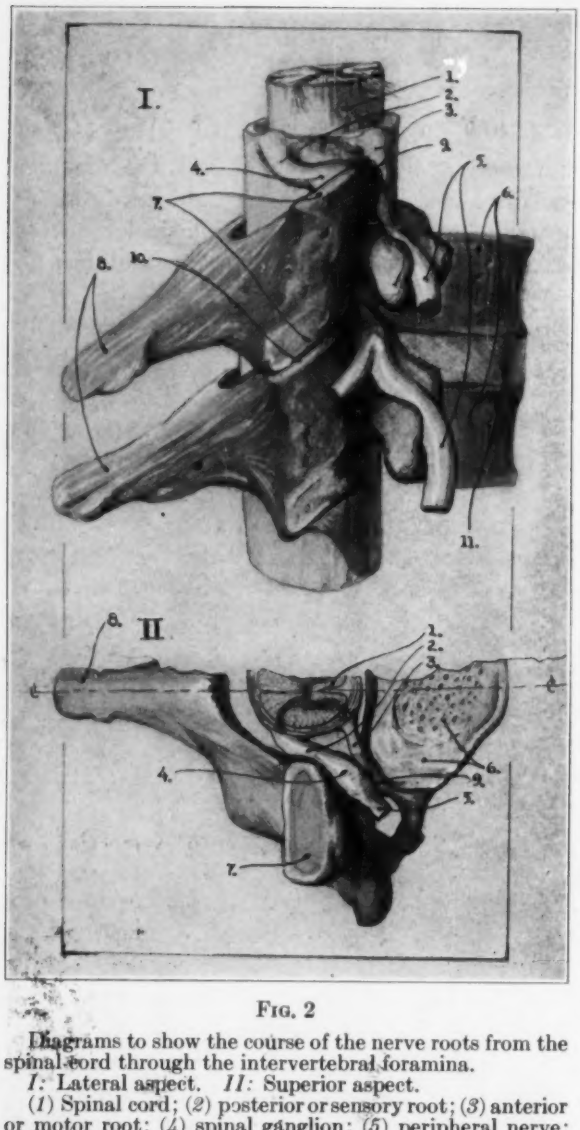


FIG. 2

Diagrams to show the course of the nerve roots from the spinal cord through the intervertebral foramina.

I: Lateral aspect. *II:* Superior aspect.

(*1*) Spinal cord; (*2*) posterior or sensory root; (*3*) anterior or motor root; (*4*) spinal ganglion; (*5*) peripheral nerve; (*6*) body of vertebra; (*7*) articular process; (*8*) spinous process; (*9*) intervertebral foramen; (*10*) intervertebral articulation; and (*11*) intervertebral disc.

because of the movement that takes place at these articulations. Since the nerve tracts pass by these points of movement, derangements of the articulating structures may prove to be mechanical irritants to the nerve tract. At the occipital-atlas joint, this is represented clinically by platybasia, a condition which, though rare, does exist. On the other hand, near the articulations between the various vertebrae there are many more potential sources of trouble and, because of the structural characteristics, much greater vulnerability.

It is in their passage through the intervertebral foramina that the nerves are particularly subject to irritation. Here the nerve roots emerge from the vertebral canal to the outside of the spinal column through channels formed by notches in the adjacent surfaces of contiguous vertebrae. These notches exist just in front of the articular processes and just behind the bodies of the vertebrae, and are so fitted that they make a bony tube (Fig. 2). The completion of the tube is accomplished posteriorly by the binding together of the articular processes by a capsule that encloses the joint, and anteriorly by the intervertebral disc. There is thus formed a conduit that is fairly solid, but not entirely rigid. When the neck flexes, the upper and lower walls of the notches are pushed closer together because the hinge action is just behind them (Fig. 3). When the neck is extended, the bony portions of the foramen are separated and so the foramen is enlarged. The posterior wall of this tube consists in great part of the capsule, so that when the capsule is swollen or distended by intra-articular fluid there is a protrusion into the lumen. Also, if the intervertebral disc in the front part of the tube should be compressed, so as to produce a herniation posterolaterally, there must be a reduction in the size of the passage for the nerve. Under normal conditions, there is adequate room for the nerve to run through the foramen; but it can be seen that, when abnormal conditions exist, compression of the nerve root can easily be produced in this region.

It is interesting to note that, within the relative protection of the intervertebral foramen, the nerve structure is such that there is particular susceptibility to irritation of the *sensory* neurons. Here, in close approximation to the capsule of the joint, the posterior roots lose the protective myelin sheaths of the axons and enter the spinal ganglion to join the relatively uninsulated nerve cell, coated only by a neurolemmal sheath. These

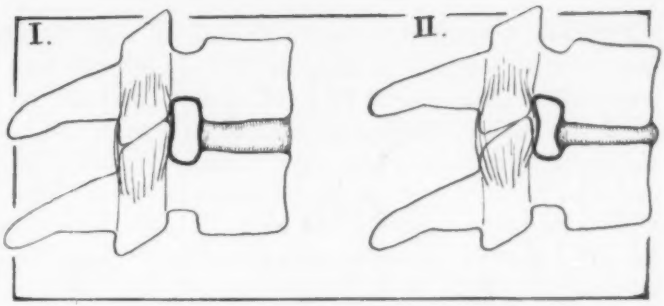


FIG. 3

Schematic diagram to indicate reduction in size of intervertebral foramen, when the neck is flexed.

cells, the first station in the afferent system, lie with their nutritive blood vessels in an areolar tissue surrounded by a thin capsule that is an expansion of the epineural sheath. This constitutes the ganglion, a structure of purely sensory cells that lies just anterior to the capsule of the joint. The motor fibers, on the other hand, enter the canal anteriorly; do not lose their sheaths; and form a much smaller cable than the sensory root with which it joins distal to the ganglion. Since the ganglion contains the cells for all types of afferent stimuli, pressure here can give any or all kinds of disturbances of sensation. This differs radically from lesions in other areas,—as for instance, near the center of the cord, where only pain and temperature discrimination would be affected. The motor nerves in this area are relatively less vulnerable. Severe compression in the foramen is necessary to produce a motor disturbance.

Thus, though pain in the arm can be produced by irritation of the afferent nerve tracts at many points, the region of the intervertebral foramina affords both the mechanism and the neural characteristics necessary to produce the classic syndrome of brachialgia. An examination of the entire clinical picture will give the basis for differential diagnostic consideration of the other conditions producing pain in the arm.

DISORDERS OF THE CERVICAL SPINAL ARTICULATIONS

The "backache" that is so often the chief complaint of our patients generally has reference to the lumbar region, but the cervical portion of the spine is likewise a frequent source of symptoms. Like the lumbar region, the upper spine has considerable mobility with the inevitable wear and tear of moving parts. Because it is more dependent on muscular support than the thoracic spine, it can easily fall into malpositions that lead to strain. It does not carry as heavy a load as the lumbar area, and does not have the same long lever in such manoeuvres as bending forward or in lifting objects, but its greater mobility and more delicate structure render it susceptible to similar stress changes. In the lumbar spine the lower part of the lordotic curve affords the most vulnerable area. In the cervical spine it is likewise the anterior curve that is most susceptible to wear. The flexibility of this region brings with it inevitable loss of stability, so that sprains in the articulation of the neck are not uncommon. The relatively fragile structure of this part of the spine accounts for the frequency of fractures and dislocations. The pathology produced varies with the particular etiological factor. Synovitis, with increased articular fluid, or capsular thickening may result from rheumatoid arthritis. Degenerative changes may produce spurring of the articular edges with bony excrescences bulging the capsule into the foramen. Osteophytes may protrude into the lumen from the bodies of the vertebrae (Figs. 4-A and 4-B). Sprains, with the tearing of the capsule, produce swellings like those seen in more exposed joints. Fractures of the vertebral arches can produce immediate displacements that cause protrusions into the intervertebral foramina. Dislocation may cause almost an obliteration of these canals. In all of these conditions there is likely to ensue a reduction in the lumen of the foramen.

PRESENT ILLNESS

Patients present themselves with one of a variety of complaints which have as their common denominator the brachialgia. There may have been a long automobile drive

with the driver sitting in a slumped position, with his head carried forward; or the patient may be a student or stenographer sitting for many hours with the head hanging over his work. The older person may tell of a gradual loss of height, with fixed sagging of the head and neck. At times an individual is met who has been suffering from chronic arthritis, polyarticular in character, with involvement of first one joint and then another; and who, without any particular injury or strain, begins to complain of pain in the neck with brachialgia. Others will give a history of a sudden strain. This may take the form of a sharp twist of the neck when the trunk is fixed, or a sudden bounce in a jolting automobile. Parachute jumpers may attribute this pain to the jar associated with the sudden opening of the parachute, while they are falling in an awkward position. Several patients have told of getting a sudden kink while drying their backs after bathing. Finally, there are the falling accidents,—catapulting out of an automobile onto the head or shoulder, or falling down a flight of stairs, or being dragged when landing horizontally in a parachute jump. Most of these people will complain of pain in the neck, shoulder, and arm. As a rule the symptoms are unilateral. Pain may radiate down to the fingers. Some have only arm pains without much neckache. Others will tell of pain radiating down the back or chest lateral to the spine. Some of these patients believed they had heart trouble, because they had pain in the precordium; or broken ribs because there was pain in the thoracic cage. Some women with this condition seek medical advice for a supposed cancer of the breast, because of the pains referred to the anterior chest wall. At times, the complaints include pain in the upper neck with radiations behind the ear along the course of the great occipital nerve. The pains may be described as an ache, a sharp stabbing pain, a numbness, or a burning feeling. They are frequently exaggerated by changes in position and are often particularly bad when the patient tries to read in

FIG. 4-A

FIG. 4-B

Roentgenograms of the cervical spine (touched up at the sixth and seventh cervical vertebrae to increase the contrast).
(*a*): Articular spurs projecting into the intervertebral foramen;
(*b*): Articular spurs projecting anteriorly from the bodies.
Fig. 4-A: Oblique view to show collapse of the intervertebral disc.
Fig. 4-B: Lateral view.

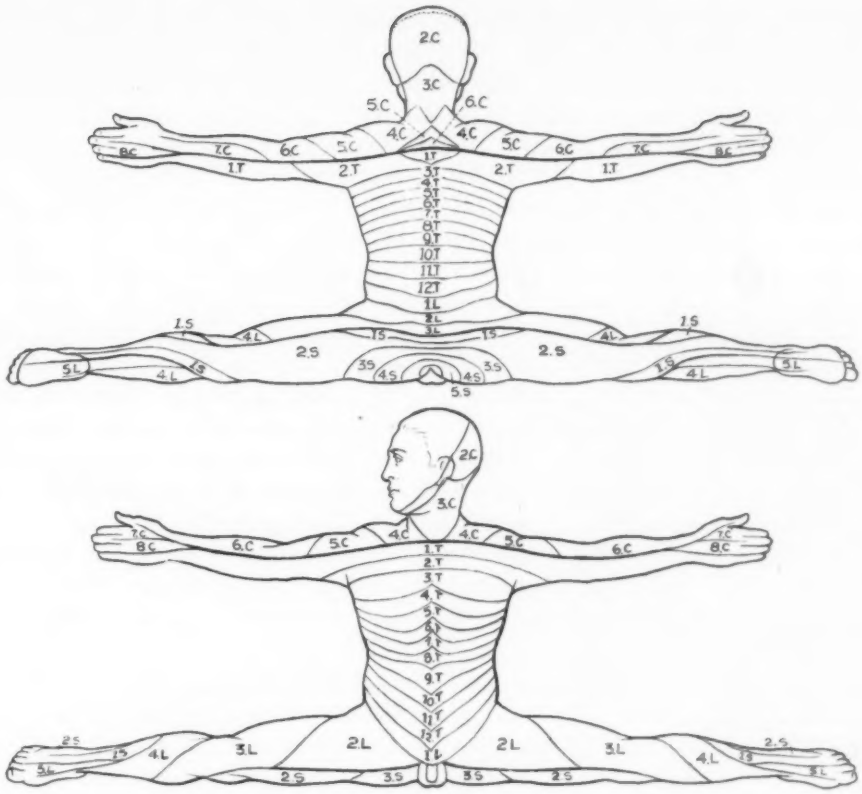


FIG. 5

Diagram to show the segmental distribution of sensation over the body. *(Reproduced from The Principles of Neurological Surgery. Ed. 2, p. 22, Fig. 5, Philadelphia, 1942, through the courtesy of the author, Loyal Davis, and the publisher, Lea & Febiger.)*

bed with his head supported on one arm. Weather changes and fatigue may be aggravating factors.

PHYSICAL EXAMINATION

The physical examination should include a careful orthopaedic and neurological survey of the parts involved. The cervical spine is usually found flexed at the cervicothoracic angle,—that is, the head is carried forward. At times the head is deviated laterally. The muscles of the neck are spastic, particularly the trapezii and the semispinalis capitis. Movements of the neck are restricted in some or in all directions. The loss of motion may involve the entire cervical spine, but is usually limited to the lower cervical spine. Palpation of the neck reveals a number of sensitive areas. As a rule the spinous processes are not remarkable for their sensitiveness, but pressure over the articular processes reveals points of tenderness over some, while the others are free from pain. The neurological examination is generally negative objectively, but at times one may find pilomotor disturbances, segmental areas of sweating, and even diminished or lost reflexes. It is, however, of great importance to map the distribution of the subjective sensory disturbances and to determine which dermatomes are involved. The roentgenographic findings will vary with the nature of the lesion. Fractures of the spine and dislocations are readily visible. The strains and sprains in younger people do not show up roentgenographically. On the other hand, in older people one may find articular lipping to substantiate the diagnosis of arthritis. One must also look for cervical ribs and other congenital malformations such as enlarged transverse processes. It must be borne in mind that the presence of bony irregularities on the film does not necessarily

mean that the patient is suffering from these changes. Such bone changes are frequently seen without any clinical symptoms. This same observation may be made with regard to disappearance of the pulse when the arm is elevated. Many people present this phenomenon without suffering any pains in the arms.

DIFFERENTIAL DIAGNOSIS

Careful analysis of the neurological manifestations is helpful in localizing the lesion. There are several valuable considerations.

1. The skin dermatomes have been charted and give a clear picture of the nerve or nerves along which the symptoms are projected (Fig. 5).

2. Dissociation of the senses,—that is, the loss of some forms of sensation, while others remain normal—indicates that the lesion is central to the nerve roots.

3. Loss of the deep reflexes follows a break in the reflex arc. The center for the biceps reflex is in the spinal cord sections of the fifth and sixth cervical vertebrae; the triceps, in the sixth and seventh cervical; and the wrist, in the seventh and eighth.

4. If the lesion produces a concomitant flaccid paralysis, it involves the peripheral nerve, since flaccidity is characteristic of damage to the lower motor neuron.

5. If a spastic paralysis exists with the sensory changes, it may be assumed that the pathology is in the spinal cord or higher.

6. A single irritant that affects the lower limbs as well as the upper limbs is likely to be a central lesion.

7. The involvement of a number of nerve tracts, so distributed that one single lesion cannot affect them all, can be attributed to a disease process that involves more than one area,—for example, multiple sclerosis or arthritis. Naturally, the location of the pathological process determined by skeletal examination should coincide with that arrived at by the neurological analysis.

Thalamic tumors are characterized by pains of great intensity, by dissociation of the senses, and by evidences of intracranial pressure. *Platybasia* is manifested by pains and weakness in the arms, by an "onion-peel" distribution of sensory disturbances in the face, and by dissociation of senses. The roentgenogram is of positive diagnostic value. *Cord tumors* likewise give a differential pattern of sensory phenomena, and are usually associated with motor changes. These tumors may affect the lower extremities. The cerebrospinal fluid usually shows xanthochromia, increased globulin, and an increased cell count. Myelograms may show filling defects or even complete block. *Herniated discs* give considerable difficulty in differential diagnosis because (1) the nerve tract is irritated at the intervertebral foramen, and (2) because the injured disc produces changes in the functional tests of the cervical spine. It is, however, noteworthy that the disc protrusion is, as a rule, limited to only one root. Some of the herniations of a nucleus pulposus bring about a loss of reflexes, atrophy of the muscles, and positive findings on lumbar puncture. *Superior sulcus tumors* are characterized by intractable pain, and, upon roentgenographic examination, show destructive changes in the upper thoracic ribs and vertebrae. *Cardiac involvement* is usually indicated by a characteristic type of pain. The pains here are frequently substernal, while those associated with spinal lesions are always off to the side. The anginal pains are quite sharp and are associated with radiations which usually follow the inner aspect of the arm. The fear of death that so often accompanies these is not felt in the cervical-spine syndrome. Furthermore, heart lesions can generally be recognized by local physical findings in the chest and by electrocardiography.

Lesions in the supraclavicular region have received a great deal of attention. *Cervical rib* produces its irritation distal to the roots, and does not involve those nerves which branch off in the first part of the brachial plexus. Furthermore, it cannot catch the fibers that make up the cervical plexus, so that the concurrent involvement of the upper

nerves should rule out the rib as a factor. The location of the irritation in cervical rib is near the subclavian vessels, so these are often involved in the clinical picture, with resultant vascular changes in the arm and hand. *Tumors* in this region can usually be felt, and are likely to produce circulatory changes. This is also quite true of aneurysms of the vessels. Spasm of the *scalenus anterior* has been reported as an etiological factor, but this, too, if responsible, must exert its effects on the distal portion of the plexus and should not disturb the upper cervical nerves. *Shoulder injuries* sometimes produce radiations, but these are usually limited to the distribution of the circumflex nerve. Shoulder pathology is also easy to find, because of the relative superficiality of this joint and because of the ease in testing its function.

Intervertebral foraminal compression causes irritation of the ganglion, and, in more severe cases, of the motor fibers. The manifestations are, therefore, essentially sensory in most instances. Furthermore, the disturbances are characterized chiefly by paraesthesia and do not show any dissociation of the senses. Because there are conditions, such as arthritis, which affect more than one intervertebral foramen, nerve-root irritation can be found at a number of levels. Indeed, it is not uncommon to find a cervicalgia that has symptoms referred along the cervical plexus, concomitant with the brachialgia. Furthermore, the intervertebral foraminal irritation can produce radiations to the posterior and anterior chest walls. Above all, it must be stressed that, in brachialgia of intervertebral foraminal origin, evidences of involvement of the spine must be found, and that the particular part of the spine involved must coincide with the course of the nerves down which the radiations are projected.

FIG. 6

Strapping to hold the cervical spine in extension.

Having determined the location of the pathology, the nature of the lesion must next be considered. The intervertebral foraminal areas present the greatest variations in types of pathology. Traumatic lesions usually have a history which cannot escape attention. Compression fractures of the bodies of the vertebrae frequently have associated manifestations of a compressed cord, and are easily recognized by routine roentgenograms. It is the fracture of pedicle, articular process, or lamina that must be searched for. Careful roentgenograms, taken not only in the anteroposterior and lateral views, but also in the oblique exposures, are necessary. Dislocations can usually be seen in the lateral views. Sprains are usually diagnosed on the basis of sudden injury without roentgenographic evidence of bone damage. Strains are associated with malposture, coupled with the added stress of prolonged activity in an exaggerated malposition. In arthritics, there is, as a rule, no history of sudden injury; the onset is insidious; there may be polyarticular manifestations; pains are generally worse on first arising in the morning; and frequently there is an aggravation of discomfort when the weather changes. In older people, roentgenographic changes consisting of arthritic spurring are usually noted.

TREATMENT

Treatment must, of course, be directed along etiological lines. The tumors, whether they be in the brain, spinal cord, the superior sulcus, or in the supraclavicular region, are treated on their own merit. As far as the brachialgia is concerned, removal of the pressure is usually all that is necessary. Platybasia can be relieved by decompression. Cardiac involvement naturally calls for treatment of the specific type of pathology. Herniated discs are at the present time being treated by exploratory operation and the evacu-

ation of the degenerated nucleus pulposus. Fractures and dislocations of the spine require reduction and immobilization. For sprains, immobilization alone is indicated. Strains call for the avoidance of malposture and changes in the occupational habits that lead to this. If muscle contractures have already taken place, heat and massage, followed by a course in postural rehabilitation, should prove helpful. Arthritics must be combed for foci of infection; and, if possible, any that are found should be removed. In osteo-arthritics, physical therapy is usually helpful. But in all of these, attention should be directed to the avoidance of pressure in the intervertebral foramina. Since hyperextension usually helps enlarge these, it is desirable to institute this as early as possible. In the milder cases it is enough to explain to the patient the mechanism producing his brachialgia and to teach him how to acquire proper posture. Here it must be mentioned that tilting the head up so that the nose points upward does not yield hyperextension in the lower cervical spine. Indeed, this manoeuvre is likely to increase flexion in the area. Instruction should stress the fact that it is necessary to keep the chin down while retracting the head, so that the weight of the head rests directly over the shoulders. When the patients cannot or will not remember this, strapping will prove helpful (Fig. 6). With a strapping that grasps the nape of the neck and holds it back by adhesive, the patient can be comfortable as long as he keeps his neck properly aligned; but if he allows his head to slump forward, the tug of the adhesive will prove an uncomfortable reminder. Sleeping on the back without the use of a pillow should be encouraged. When the patient must lie on his side, a pillow should be packed under his head so that the right angle made by the head and neck with the shoulder girdle is maintained. It is often helpful to have the patient lie supine and to pack sandbags on either side of the head to prevent movement. In more severe or more resistant cases, head traction is usually of tremendous help. This may have to be followed by immobilization in the corrected position. For this a Thomas collar, a brace with a chin support, or even a Calot jacket may be found necessary. In all of these procedures, the important thing is the maintenance of hyperextension of the lower cervical spine.

SUMMARY

To summarize, brachialgia, which means pain in the arm, is a symptom complex. It can be produced by any one of a number of lesions affecting the sensory tracts supplying the arm. The site of the lesion can be traced by following the distribution and the character of the sensory changes. Clinical experience reveals that by far the most frequent location for the nerve irritation is in the intervertebral foramen, and anatomical and physiological considerations clarify the relative ease with which sensory disturbances can be produced here. Affections of the intervertebral foramina are usually associated with clinical evidences of pathology in the cervical spine. Treatment must be directed to the primary etiological factor, but hyperextension of the spine will tend to reduce the compression of the nerves within the intervertebral foramina.

A PRACTICAL TECHNIQUE FOR PRESERVING SURGICAL AND ANATOMICAL DISSECTIONS *

BY J. B. DEC. M. SAUNDERS, M.B., CH.B., F.R.C.S. (EDIN.), AND
A. H. RICE, M.D., SAN FRANCISCO, CALIFORNIA

From the Division of Anatomy, University of California Medical School, San Francisco

This year we celebrate the four-hundredth anniversary of the publication of that great masterpiece, the *Fabrica de Corporis Humani* of Andreas Vesalius, the fount from which the modern science of anatomy sprang. Four hundred fruitful and productive years have culminated in the present, when, once again, the exigencies of war have re-emphasized the importance of anatomy as fundamental in the basic training of the surgeon. Yet, there has been perhaps no period in this long history when recrimination and criticism over the deficiencies in the teaching of this important subject have been more marked between clinician and anatomist. How serious are these deficiencies, even in postgraduate training in the specialties, is apparent from the recent report by Whipple of the results of the examinations in anatomy by the American Board of Surgery.

This criticism, both just and unjust, stems without question from a failure to recognize that the study of medicine in all and any of its aspects requires the development in our schools of a unified and coordinated point of view in its several departments, for only in this way can existing barriers between such departments be broken down. The future physician or postgraduate comes to an institution to be trained in medicine or surgery and not in anatomy, biochemistry, physiology, pathology, *et cetera*. Nonetheless, it is important to remember that to serve, and serve only, the purely pragmatic needs of the student is to stunt progress, and will lead only to decline. The careful and sympathetic blending of the academic and the practical, by nourishing the delicate flower of initiative, and by mutually receiving nurture and inspiration from the countless problems of the clinic and of the laboratory, offers opportunity to both student and preceptor to participate in a progressive ideal. It was with such considerations in mind that the Division of Anatomy participated in the program of training in orthopaedic surgery at the University of California under the direction of Dr. LeRoy Abbott. In order to bridge the gap between the ancillary sciences and clinical work, this Division for the past ten years has seen to it that an adequate liaison has been maintained. Unless some of its members are given opportunity to become familiar with the clinic, a sense of proportion cannot be developed in the early years of instruction. This relationship should continue throughout the curriculum into the postgraduate years, with changing emphasis on its applied aspects.

Any such conception needs more than a plan on paper or a series of didactic lectures, for, to become a *de facto* accomplishment, it requires the creation of facilities and opportunities; so that it will not degenerate into another method of feeding pap, instead of becoming a source of inspiration.

As part of the plan in orthopaedic instruction, we have established what we prefer to call workrooms or a laboratory, rather than a museum. These contain preparations of value in this specialty, which the student may consult at any time on his own initiative. Here, biological and functional as well as the morphological aspects are represented, for, not only are the cadaver and wet specimens available for the solution of problems of technique and approach, but, for example, the large and important subject of bone is represented by histological and other preparations to illustrate its development and its response in health and in disease. Here, too, seminars in fundamental aspects are arranged in the presence of illustrative material, and research is encouraged.

* Read at the Annual Meeting of The American Orthopaedic Association, Cleveland, Ohio, June 7, 1943.

One of the difficulties is that time is seldom sufficient, especially in later years, to allow the student, hard-pressed resident, or busy surgeon to refresh his mind on some anatomical point or surgical approach by actual dissection. For this reason, and because the wet specimens are messy and deteriorate rapidly, we developed, several years ago, a simple, cheap, and effective method of permanently preserving interesting or instructive dissections, with sharp and accurate differentiation of structures. We have found these specimens particularly useful for illustrating orthopaedic procedures. Clean, odorless, and relatively indestructible with reasonable care, they are appropriate in the classroom, and can be loaned to the student in much the same way as a library loans its books. We have found them to be of tremendous value in the conduct of special courses in surgery of the extremities which the present War has demanded.

The Egyptians were undoubtedly the first to use resinous substances in the preservation of human bodies. Their mummification method was essentially a process of evisceration, maceration in a solution of sodium carbonate, drying, and the application of resinous substances, followed by bandaging. Swammerdam, in 1670, was the first to inject and preserve anatomical dissections; but his pupil, Ruysch, to whom he taught the art, became famous through the development and the extensive application of the technique. He injected the vessels with colored mixtures of beeswax, resin, and turpentine; dissected and dried the specimens; and applied varnish to preserve them in a dry state. None of his specimens are known to exist at present. Since his day, numerous workers have advanced similar techniques. Among them, those of Bidloo, Cowper and Nicholls, Auzous, Brenetti, and Marini were the most outstanding. More recently, Souchon has carried out extensive dissections and has worked out a method of preserving them, developing his "Museum of Normal Anatomy". His method, however, requires careful selection of anatomical material (only 8 to 12 per cent. of dissecting-room cadavera are satisfactory); calls exclusively for bodies prepared with arsenic; and necessitates a prolonged, complicated, preservation process, ending with the specimens in solution. In our technique, the specimens may be selected from any well-preserved body (although we prefer the arsenic-prepared); do not require lengthy "curing" processes, with frequent changes of solutions, *et cetera*; and, above all, in the final preparation, are mounted dry in permanent colors, on a solid base, so that they may be handled and studied from all angles. The final advantage that gives an unequalled sense of gratification to the meticulous dissector, is the absolute accuracy with which even fine, hairlike fibers may be brought out.

TECHNIQUE

As previously mentioned, specimens may be prepared from any well-preserved body, although the specimens prepared with arsenic are preferred, because of the better basic color of the muscles, and because of the probable greater durability. Injection of the vessels with colored nitrocellulose, paraffin, or other material is advisable, but not necessary; it gives a better contour, particularly to the veins.

The first step in preparation is a careful, thorough, painstaking dissection of the whole specimen, with all fascia and fat cleaned away from the structures to be demonstrated, except where it is necessary that they serve as a bed for important structures. The finest nerve fibers and tiny arteries may be carefully separated and preserved, even using the dissecting microscope, where desired, since utilization of our technique permits them to be sharply brought out. In order to make the specimen of as much value as possible, all the important structures in the level being dissected should be freed and elevated from their beds. It is possible to show structures in several levels, but this makes coloring more difficult. It is advisable to leave a margin of skin around the dissected area, with margins of each layer that has been passed through extending somewhat beyond it. This helps to demarcate and orient the specimen.

Between periods of dissection the specimen may be kept in a preservative solution or

may be wrapped in moist cloths. The former is preferred, as tiny nerve fibers tend to dry and shrink easily when elevated from their beds. When the dissection is complete, the specimen is allowed to dry slowly for about forty-eight hours. This is accomplished by placing it between layers of a slightly damp (not moist) bath towel, at room temperature. When the muscles and small elevated structures begin to turn brown, and mild pressure on a mass of muscle fails to express free moisture, the specimen is ready to paint.

In our first preparations, we attempted to use colored lacquers for this purpose, as recommended by Looney. They proved satisfactory for larger vessels and nerves, but were very inadequate for the demonstration of fine vessels and nerve fibers dissected by microscope, such as the nasociliary. The colors tended to blur and run into the general lacquer coats when they were applied.

We now employ ordinary water-color poster paints, and apply them directly to the partially dried specimens, using the customary red for arteries, blue for veins, and yellow for nerves. If straight tap water is used to mix the paints, they tend to crack when drying and to separate from the painted structures. This has been obviated by adding about one drop of ordinary LePage's mucilage to every two drops of water used in mixing the paint. This also tends to give a thicker paint which fills in minor irregularities and provides some adhesive quality where tiny nerves or vessels have been broken. Mucous membranes are painted to simulate the natural shade. Muscles and glands are better not painted with water colors. Ordinary brown blackboard chalk, applied directly to the muscle, with a touch of red, gives a satisfactory color, while glands are tinted with a mixture of powdered chalk to simulate the normal appearance.

As a rule, the muscles and glands are chalked first; then the other structures are carefully painted in order of their depth, the deeper ones first. Pins are used freely to retract more superficial structures while the deeper ones are being painted. We employ short sections of glass rod to keep overlying structures from touching those painted, and to keep painted structures elevated from their bed until dry. The duration of this process varies, of course, with the size and intricacy of the dissection. It may be necessary to allow a whole level to dry before resuming the painting. Misplaced drops of paint may be removed by touching the spot lightly with a large moist brush. The size of the brush employed will vary with the size of the structure being painted, and the brushes should be preserved carefully, and cleansed in plenty of fresh water between colors and after using.

After the painted areas have dried, all pins and glass rods may be removed and retracted vessels and nerves returned to their normal positions.

Preservation is started by spraying the dissected area from several angles with a mixture consisting of one part clear commercial lacquer and two parts amyl acetate. Two coats are applied, an ordinary atomizer being satisfactory for use in spraying. Straight amyl acetate should always be run through the atomizer after use to prevent clogging. The larger masses of tissue, including large muscles, are then thoroughly infiltrated with absolute alcohol by means of a Luer syringe, injection being done from several angles, and care being taken to carry the injection clear to the bone, while the surfaces are kept flooded with the same fluid. This is permitted to dry for twenty to forty minutes, and the process is repeated with amyl acetate. After drying for another forty minutes, the structures are well infiltrated with a mixture consisting of one part clear lacquer and two parts amyl acetate, and allowed to dry again for one hour. A mixture of one part lacquer and one part amyl acetate is then sprayed over the whole specimen from several angles, care being taken to get the coat all around the small vessels and nerves. Two such coats are applied, each being allowed to dry.

If the preparation contains large and bulky masses of muscle and soft tissue in which considerable shrinkage would be anticipated, this can be controlled and the shape of muscle masses preserved by the use of a filler. We have found that plastic wood, forced into and distributed through the muscle mass, is excellent for this purpose.

The specimen is now ready for mounting. The body is trimmed off evenly, and pieces of cork are firmly attached by means of wood screws to convenient bony areas. We have found that shrinkage is well taken up by inserting wooden pegs, two by two by fifteen millimeters in size, between the skin and subcutaneous structures at one-half-centimeter intervals all around the skin margin which is to be inserted in the base. A rigid mold is prepared, the size and shape of the base desired, and the specimen is placed in the proper position.

After trying various substances as material for the base, we found that it was necessary to use some type of dental plaster, such as the proprietary mixture "hydrocol". This is quite inexpensive and is much harder than plaster-of-Paris, which has been found entirely unsatisfactory, since it absorbs fatty material from the specimen and softens. The dental plaster is mixed with water, and, with the specimen in place, is poured into the mold to the proper level and allowed to harden.

While the plaster is drying, the dissected parts are covered with two coats of pure lacquer, completely surrounding the dissected vessels and nerves, and each coat is allowed to dry thirty minutes. This is best accomplished with a brush or a regular pressure lacquer sprayer. The base is now labeled in India ink, allowed to dry thoroughly, and covered with two coats of pure lacquer. When these have dried, the specimen is finished. Like all organic material, the preparations must be protected from moths.

SUMMARY

A technique is presented whereby surgical and anatomical dissections may be preserved in dry, odorless, permanent preparations, demonstrating the most minute detail in sharp contrast. It includes:

1. Careful, clean dissection;
2. Painting with water colors;
3. Preservation of tissues by infiltration with absolute alcohol, amyl acetate, and lacquer.

We should like to acknowledge the services of Mr. Rudolph Skarda, whose painstaking care is largely responsible for the success of the method.

REFERENCES

FIFE, ANDREW: A Compendium of Anatomy of the Human Body. Vol. I. Philadelphia, 1802.

GARRISON, F. H.: An Introduction to the History of Medicine. With Medical Chronology, Suggestions for Study and Bibliographic Data. Ed. 3. p. 248. Philadelphia, W. B. Saunders Company, 1921.

KEEN, W. W.: A Sketch of the Early History of Practical Anatomy. The Introductory Address at the Philadelphia School of Anatomy. Philadelphia, J. B. Lippincott & Co., 1874.

LOONEY, W. W.: An Improved Method for Painting Dissections. Anat. Rec., XXXVIII, 241, 1928.

PARSONS, USHER: Directions for Making Anatomical Preparations, Formed on the Basis of Pole, Marjolin and Breschet, and Including the New Method of Mr. Swan. Philadelphia, Carey & Lea, 1831.

SOUCHON, EDMOND: The Preservation of Dissections of Surgical Anatomy with Permanent Color of Muscles, Vessels, Nerves, and Organs, by a New Method. Surg. Gynec. Obstet., IX, 25, 1909.

Preservation of Anatomic Dissections with Permanent Color of Muscles, Vessels and Organs by Newer Methods. Anat. Rec., X, 43, 1915.

Preservation of Anatomic Dissections with Permanent Color of Muscles, Vessels and Organs. A Supplementary Note, Describing Another Method, the Curing Method. Anat. Rec., XII, 313, 1917.

The Souchon Museum of Normal Anatomy. *In* International Assn. of Medical Museums. Bull. VII, 42, 1918.

SYMPATHETIC BLOCK IN THE TREATMENT OF LOCAL SHOCK *

An Experimental Study

BY JAMES E. M. THOMSON, M.D., LINCOLN, NEBRASKA, FERDINAND HELWIG, M.D., KANSAS CITY, MISSOURI, AND EUGENE SIRE, B.S., LINCOLN, NEBRASKA

"Local shock" is a term applied to a clinical picture, often exhibited after severe injuries (particularly crushing injuries) of the extremities. It is characterized by evidence of local traumatic vasospasm in the region of injury, which may extend to involve a considerable part of the extremity. "Local shock" is to the extremity what general shock is to the total human organism. This phenomenon may at first act as a defense or safety reflex on the part of nature to prevent hemorrhage and further trauma to the region. On the other hand, if the "local shock" continues for a sufficient period of time, capillary incompetency or decompensation in the area results in interference with the normal interchange of tissue fluids and a piling up of the products of trauma, thereby ending in degeneration and local death of tissue before circulatory integrity can be re-established. To combat this process, various measures have been developed. Among them, blocking of the vasoconstrictor apparatus to the extremity with novocain has been suggested, and apparently has been successful. In many instances, it has been felt that this procedure was eminently justified. Apparently the circulation to the part is thus revived, and an early healing process is stimulated.

To evaluate the importance or influence of sympathetic block on this state of "local shock", the following experimental project was undertaken. A preliminary report of this work was made at the Annual Meeting of The American Academy of Orthopaedic Surgeons.† While considerable work has been done since then, the problem offers many ramifications that have not yet been investigated.

This is a report of the objective physical and histopathological observations made on over 100 experimental animals (albino rabbits), which were sacrificed 100 hours after severe crush fractures of the right humerus in the mid-shaft. Four hours after the injury, one-half of these animals received an injection of three cubic centimeters of novocain hydrochloride (2 per cent.) into the region of the right stellate ganglion. The remainder of the animals were used as controls and had no treatment. All of them were observed regularly at frequent intervals by the same person (Sire), who recorded changes in temperature of both the right and left upper extremities, and evidence of swelling. Finally, at the end of 100 hours, the animals were sacrificed; the right upper extremities were amputated at the shoulder; and sections were taken of the tissue in the region of greatest injury, at as near the same anatomical place in each case as was humanly possible. The histopathological changes were recorded (Helwig), without knowledge of whether the specimen came from a treated or control animal.

PROCEDURE

1. The albino rabbit was chosen because the lack of pigment in the eye and ear made changes in the appearance of the blood vessels readily discernible. They were selected from the same stock, were two to three months old, and were within 500 grams of the same weight, averaging slightly over 1600 grams. The stellate ganglion can be readily located in this animal with a hypodermic needle, one and one-half inches long. This body controls the vasoconstrictor mechanism of the upper extremity, as well as that of the eye

* Read at the Annual Meeting of The American Orthopaedic Association, Cleveland, Ohio, June 8, 1943.

† Presented at the Annual Meeting of The American Academy of Orthopaedic Surgeons, Chicago, Illinois, January 1943, but not published.

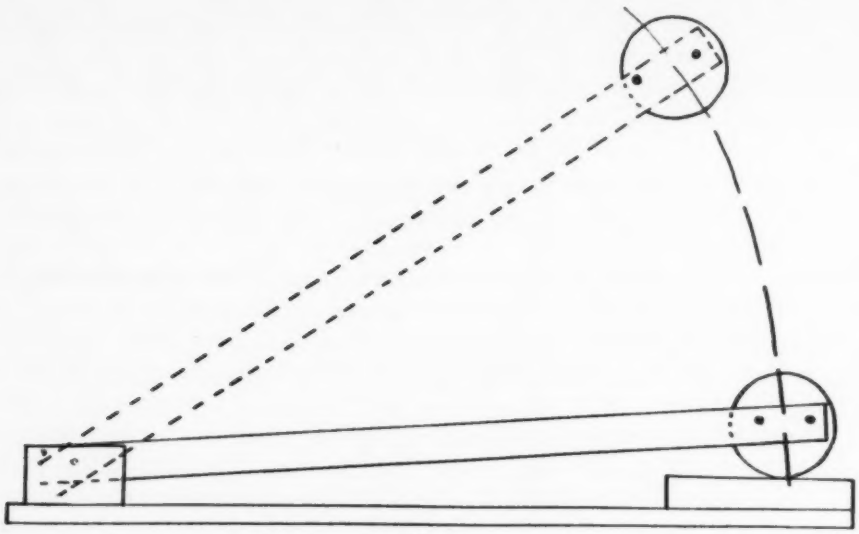


FIG. 1

Instrument for producing fracture.

and ear of the same side. The effect of a successful injection of novocain into this body is manifest by visible dilatation of the blood vessels of the eye and ear.

2. An instrument (Fig. 1) was devised that would consistently deliver the same type of crush fracture with equal violence and equal injury to the soft tissue. Since a falling body of uniform contour will always travel at the same rate of acceleration, the apparatus was designed with this in view. A hard maple bar 97.0 by 2 by 2.5 centimeters was fitted with a pivot on a quarter-inch bolt. A circular weight, fourteen centimeters in diameter and six millimeters thick, with a rounded edge, was bolted with its center ninety centimeters from the center of the pivot bolt. This entire assembly was fastened to a baseboard 107 by 2 by 20 centimeters. An anvil block, four centimeters thick and twenty-four centimeters square, was attached to the baseboard in the proper position for the hammer of the apparatus to strike. A release was fixed to a standard beside the pivoted arm to hold the hammer in the raised position, and to release it to fall with a minimum of friction.

By a few simple experiments, in which the weight was allowed to fall upon the humerus of an anaesthetized rabbit, it was determined that a fall of forty-five centimeters produced a fracture which proved to be very uniform when examined roentgenographically (Fig. 2).

When computed, the force of the energy expended was found to be 3.94 joules, or 2.90 foot-pounds. Calculations are as follows:

$$\begin{aligned} \textit{Loss of P. E.} &= mgh + m'gh' \\ &= (827 \times 980 \times 45) + (129 \times 980 \times 22.5) \\ &= 36{,}470{,}700 + 2{,}844{,}450 \\ &= 39{,}315{,}150 \textit{ ergs} \\ &= 3.93 \textit{ joules} \\ &= \frac{3.93 \times 550}{746} \\ &= 2.90 \textit{ foot-pounds} \end{aligned}$$

The animal was held in place in such a position that the striking surface of the weight fell on the central portion of the humerus. The right foreleg was chosen for experiment because of its convenience. All animals were given a light ether anaesthesia prior to the fracture procedure.

3. A 2-per-cent. solution of novocain, standardized commercially by a reputable pharmaceutical firm, was used. Three cubic centimeters was successfully injected into the right stellate ganglion region four hours after the crush fracture. No epinephrine was used, because of its local vasoconstrictive reaction.

The rabbit's hind legs were held by the assistant while the technician making the injection held the left foreleg and head of the rabbit in his left hand. This left the right hand free for making the injection. The needle was introduced one centimeter above and at a 90-degree angle to the central section of the right clavicle. The needle was advanced through the soft tissue until the spinous process of the first thoracic vertebra was felt. The needle was withdrawn about one millimeter and the injection was completed. This placed the injection in the peak of the thoracic cavity and just posterior to the fascia lining the dorsal wall of the thorax, which has been found to be the approximate location of the stellate ganglion.

General symptoms of stellate block are dilatation of ear vessels and eye pupils, and an increase in temperature of the foreleg of the injected side.

4. The temperature of the forelegs of the animals was taken at regular intervals by placing the tip of the thermocouple electrode in contact with the lateral surface of the foreleg at the elbow joint, and holding it in place until no more fluctuation of the needle on the scale occurred. Corresponding right and left temperatures were taken, one immediately preceding the other. The thermocouple was standardized before each set of readings was taken.

The thermocouple used gave scale readings in degrees 55 to 102 Fahrenheit and 13 to 41 centigrade.

The normal temperature of the rabbit varies considerably over a twenty-four hour period, and is distinctly influenced by room temperature. Therefore, the room temperature was maintained by thermostatic control at as near 70 degrees Fahrenheit as possible.

5. The evidence of swelling in the injured extremity was noted at regular intervals

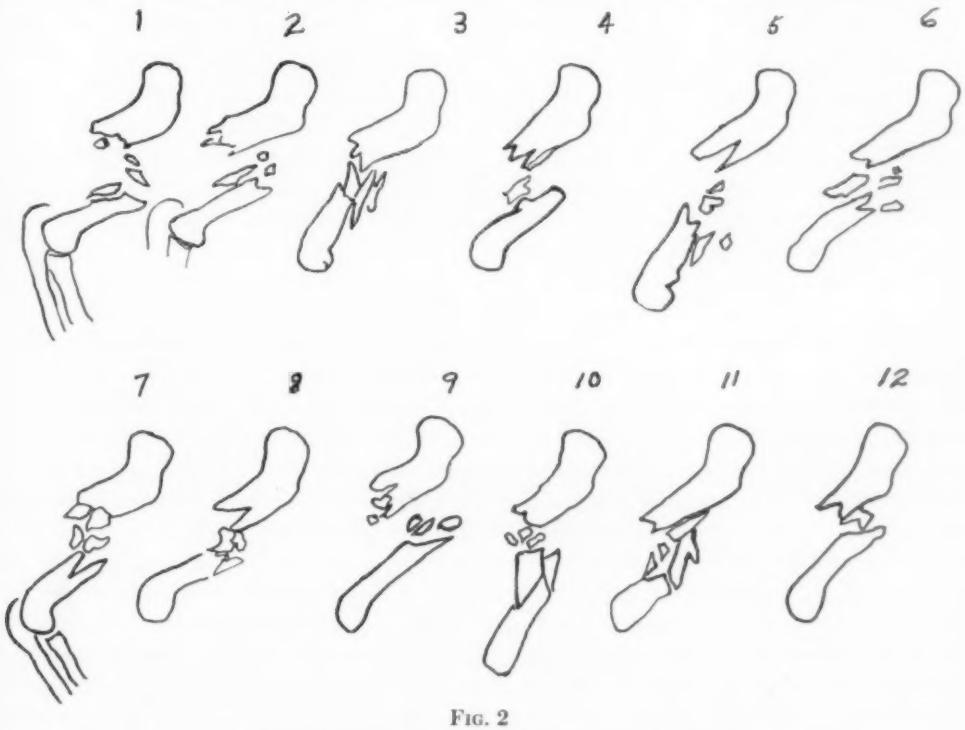


FIG. 2

Tracings of roentgenograms of both groups of animals.

CUBITAL TEMPERATURE VARIATION IN TREATED AND UNTREATED ANIMALS

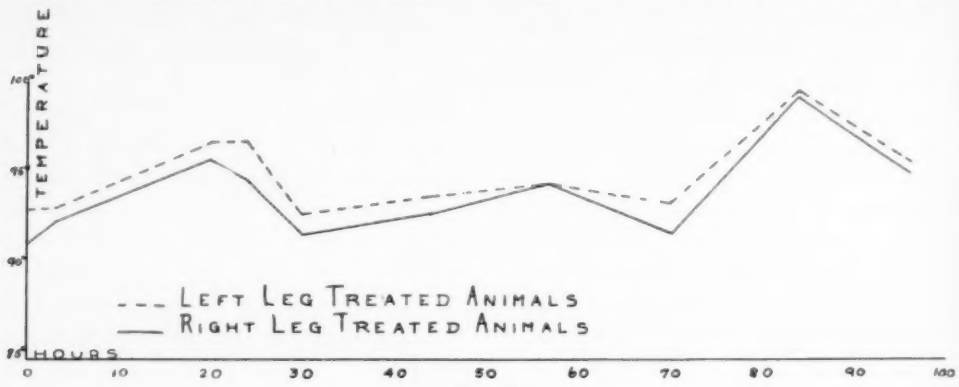


FIG. 3-A

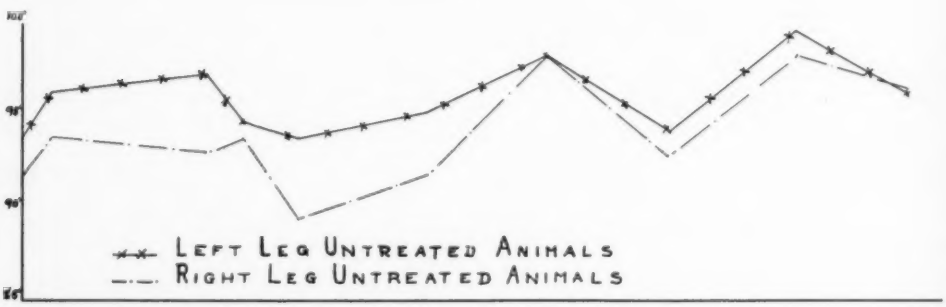


FIG. 3-B

in several groups of these animals by the same observer who was without knowledge as to which animals had received stellate block. The observation was recorded in the following terms: "great swelling", "medium swelling", "slight swelling", and "no swelling". It is realized that there is a distinct human frailty in such an observation, but when two other observers have relatively the same findings and deductions, it is more than a coincidence.

6. One hundred hours after the injury, the animals were sacrificed. Immediately after amputation of the injured upper extremity, including the scapula, the specimen was placed in formalin solution, sections were made, the slides were studied without identification, and observations were tabulated.

DISCUSSION

In the following discussion "treated animals" are those having had their right humerus fractured and an injection of three centimeters of 2-per-cent. novocain in the region of the stellate ganglion four hours after the crush injury. "Untreated animals" represent an equal number having had a similar fracture of the right humerus, but no injection.

Figure 3-A is a composite temperature chart of the treated animals. The solid line is that of the fractured right leg; the broken line that of the normal left leg. These lines run a relatively parallel course. The injured extremity is about 1 degree cooler than the uninjured extremity until about fifty-seven hours have elapsed; then the lines converge, but again promptly return to their relatively parallel position. At eighty-four hours they reach a peak rise of temperature, but turn downward immediately.

Figure 3-B is a composite temperature chart of the untreated control animals. The lower interrupted line portrays the temperature of the fractured right leg, and the "X" line that of the normal left leg. Here a wide variation is found in the temperatures of the two extremities. The injured right leg, at twenty hours after injury, is 4 degrees

CUBITAL TEMPERATURE VARIATION IN TREATED AND UNTREATED ANIMALS

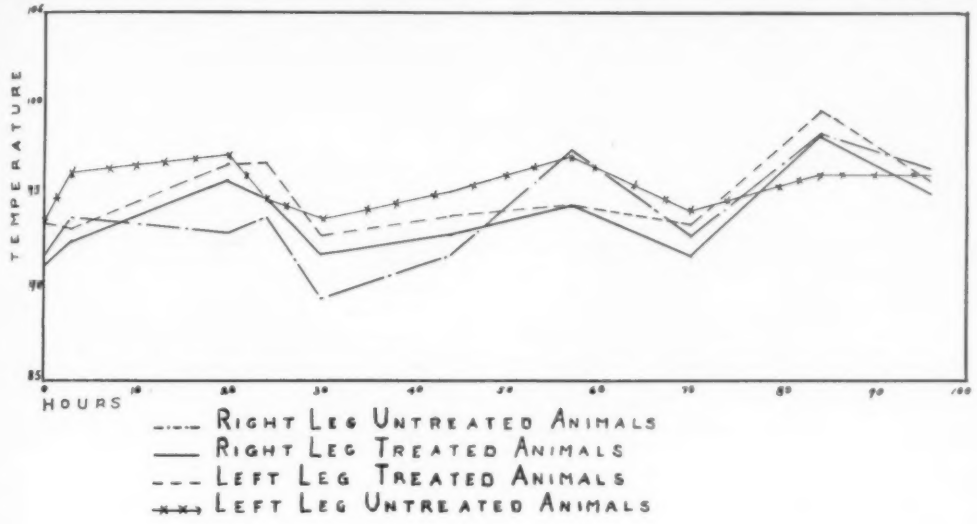


FIG. 4

(Fahrenheit) lower than the normal left leg; and, at thirty hours, it is nearly 5 degrees lower. At forty-five hours, these lines tend to converge, and they reach an equal peak temperature at fifty-seven hours; then they tend to become parallel to a higher peak at eighty-four hours, gradually receding and converging just before being sacrificed.

A comparison of these two charts indicates that the treated animals, those having a novocain block of the right stellate, had relatively the same temperature curve throughout the 100-hour period of observation, the injured right extremity being slightly cooler than the normal left. On the other hand, those which did not have the sympathetic block showed a wide divergence in the temperature curves of the fractured and normal legs, until that point (fifty-seven hours) at which both groups made a peak rise in temperature. Later, the curves tended to run parallel and converge (Fig. 4).

It might be hypothesized from these observations that the novocain block of the stellate ganglion, on the side that received the severe injury, revived the circulation of the injured extremity to nearly normal integrity, while the untreated animals suffered severe vasospasm until an inflammatory reaction brought the temperature to the fifty-seven and eighty-four hour peaks. Apparently the temperature of the treated animals continued more nearly normal. Even the normal left side of the treated animal did not have so high an average temperature as the normal left side of the untreated animal.

In a group of twenty-four animals, a second injection of novocain was made into the right stellate ganglion after forty-five hours, since that was the time when something unexplainable began to happen which caused a converging rise in temperature. However, this did not influence the curve, except to slow the rise of temperature of the right leg.

The estimation of the degree of swelling in a rabbit's extremities should be a very technical procedure. The several types of mechanical apparatus which were devised gave inaccurate and inconclusive deductions. However, after much practice, the authors finally endeavored to classify swelling by touch and feel. They recognize the frailty of even suggesting such a deduction, but it is remarkable how similar the estimations of "no swelling", "slight swelling", "medium swelling", and "great swelling" were on the part of two practised and one "amateur" observer. Therefore, the findings are offered in Figure 5. The shaded squares are estimations of swelling in the treated animals, and the clear squares of swelling in the control animals. Note that only a few of the treated animals had "great swelling", and in steplike manner the number increases as the degree

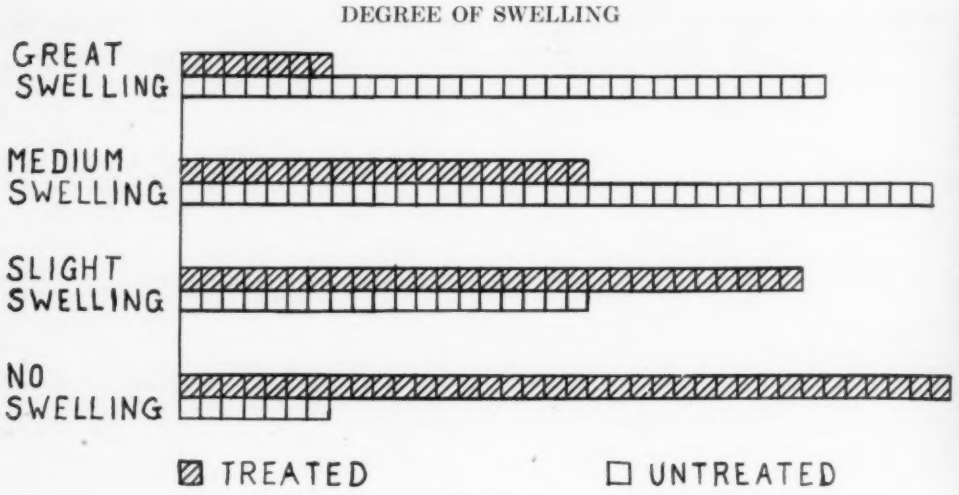


FIG. 5

of swelling decreases. Practically the opposite occurs with the untreated controls. They have the largest number in the groups marked "medium swelling" and "great swelling" and a steplike decrease in number down to the state of "no swelling". Here the hypothesis might be drawn that, with the more normal circulatory integrity brought about by blocking the vasoconstrictor mechanism of a severely injured extremity and, thereby, reducing the vasospasm, the products of the tissue trauma are carried away more efficiently by the stimulated capillary activity.

The pathologist in this experimental project (Helwig) not only cut the sections from the site of greatest injury, but examined all of the slides and made his written comments regarding each, without knowing whether the section or slide came from a "treated" or "control" animal. Each slide was judged with respect to the evidence of healing response. Those slides showing good healing response were placed in one group, those not showing good healing response were put in another group, and those which were doubtful or borderline were put in a third group.

Response to healing was judged upon the following histological findings: Excellent organization of the clot by fibroblasts and young proliferating capillaries, when abundant and overshadowing the remaining picture, was considered a good response. Furthermore, when calcification took place in muscle which was obviously necrotic, it was always found that these foci were embedded in a very active fibroblastic reaction. If there was a striking proliferation of sarcolemma nuclei, particularly when combined with fibroblastic and other fixed-tissue reactions, this was likewise considered salutary; but an abundant or predominant amount of muscle necrosis, with autolytic, non-viable, and non-calcified fibers, was considered a bad healing response. The fact that 80 per cent. of all of the sections, in the series of over 100 section slides, were placed correctly in the treated and untreated groups may be considered as more than coincidental.

A few photomicrographs, comparing the predominant histological appearance of the sections taken from the treated and untreated extremities, are illustrative of the outstanding characteristics of each.

The section shown in Figure 6-A was taken from a treated animal, and shows a most striking reaction of proliferation of fibroblasts and organization of clot. There are a few scattered, focal, calcified, necrotic fibers embedded in the fibroblastic mass. Some degenerated but viable muscle is also embedded here, the latter showing a very healthy interstitial and sarcolemma reaction. No autolytic necrotic muscle without fixed-tissue reaction is seen. This is a splendid healing response.

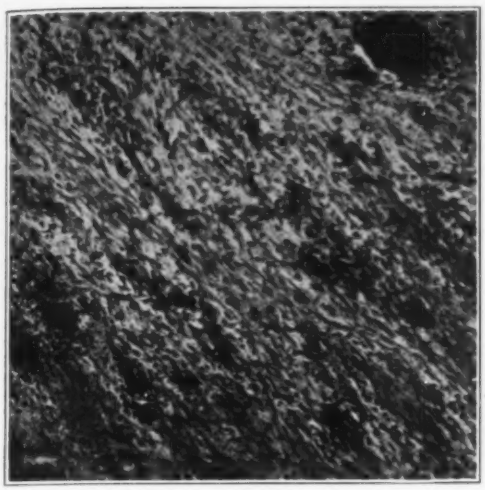


FIG. 6-A

Section from treated animal.

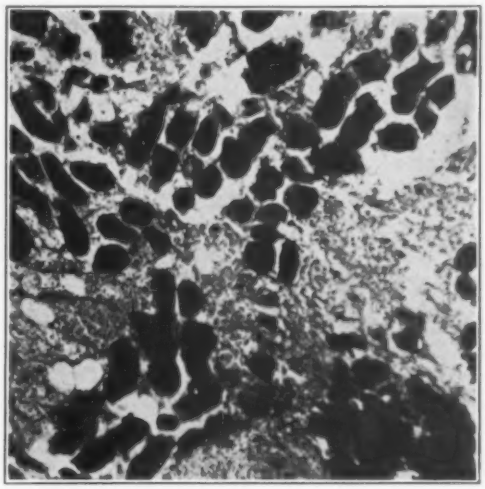


FIG. 6-B

Section from untreated animal.

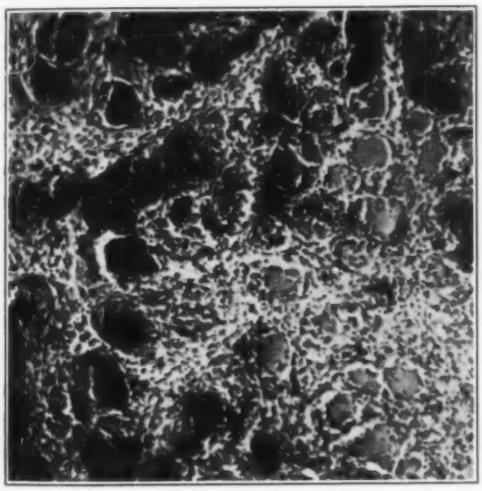


FIG. 7-A

Section from treated animal.

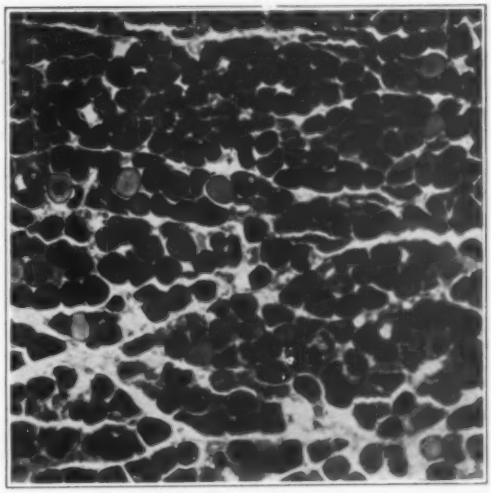


Fig. 7-B

Section from untreated animal.

The section illustrated in Figure 6-B was taken from a control animal and shows practically no healing reaction to the injury. It is composed almost exclusively of necrotic, autolyzed, non-viable muscle, in the interstices of which, aside from a few scattered leukocytes, there is absolutely no fixed-tissue or fibroblastic reaction of any character. The sarcolemma nuclei have all disappeared. There is practically no evidence of healing.

The section shown in Figure 7-A was taken from the fractured right humerus of a treated animal and shows numerous small areas of calcified dead muscle cells embedded in an unusually rich fibroblastic stroma. Focal hemorrhage with marked organization is also encountered, and large areas of viable muscle bundles are observed. Often these bundles are embedded in or infiltrated by a very rich fibroblastic reaction. Practically no aseptic, autolytic, or necrotic muscle is found.

The section shown in Figure 7-B comes from an animal in the untreated control group, and shows practically no clot organization. Some pieces of necrotic bone and autolytic muscle are embedded in one side of the clot, whereas another portion of the

FIG. 8-A

Section from treated animal.

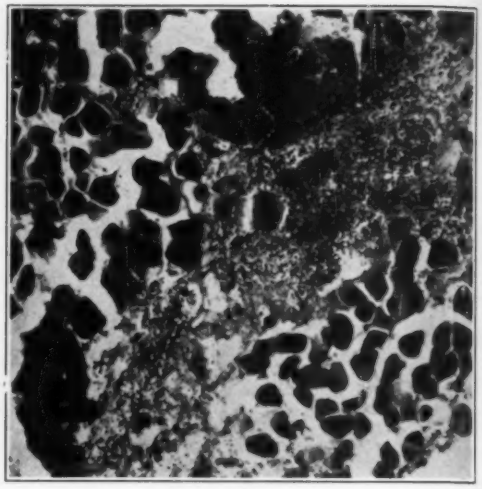


Fig. 8-B

Section from control animal.

section shows both fixed-tissue reaction and beginning clot organization. In this section is seen autolytic, necrotic, non-viable muscle bundles without sarcolemma or interstitial response.

The section illustrated in Figure 8-A was taken from the right fractured humerus of a treated animal, and shows large zones of young fibroblasts crossed frequently by proliferating capillaries which are organizing the clot. A few small autolyzed muscle bundles are seen, but there is excellent fibroblastic response around them. These autolytic fibers are very scanty. This represents an excellent healing response.

The section shown in Figure 8-B comes from a similar area of the humerus of an untreated animal. A study of this entire slide would be interesting. The histological picture is predominated overwhelmingly by fibroblastic proliferation in which focal islands of calcified, dead muscle are found. Only here and there are autolyzed muscle fibers seen without much fixed-tissue response, and there is active organization and replacement of the clot. Another section, however, is made up almost exclusively of autolyzed, necrotic, non-viable muscle surrounded by a thin zone of fibroblasts. This would be considered a poor response to healing as compared with Figure 7-A.

CONCLUSIONS

1. From these observations we conclude that novocain block of the sympathetic vasoconstricting mechanism in the severely injured extremities of experimental animals tends to restore the normal circulatory integrity to the extremity.

2. Likewise, it stimulates a more normal interchange of tissue fluids, and excites capillary activity to carry away rapidly the tissue products after trauma. Evidence of this is presented in the composite groups depicting temperature and swelling.

3. Furthermore, the histological sections verify better healing responses in the treated animals. Most of them show excellent organization of the clot by fibroblasts and young proliferating capillaries, with calcification of obviously necrotic muscle in the "treated" injured extremities; while in the untreated sections there is uniformly marked muscle necrosis with autolytic, non-viable, and non-calcified fibers.

These conclusions would seem to indicate that blocking of the vasoconstrictor sympathetic mechanism should be more frequently resorted to as an early therapeutic measure in the treatment of a severely crushed human extremity that shows evidence of "local shock", vasospasm, and similar conditions.

SHOCK IN EXTREMITY SURGERY *

BY DALLAS B. PHEMISTER, M.D., CHICAGO, ILLINOIS

From the Department of Surgery of the University of Chicago

By surgical shock is ordinarily meant the acute circulatory embarrassment or failure which is produced by accidental wounds and operations. The causes are varied and often two or more are present at the same time. The three most extensively entertained theories are the local loss of circulating fluid, toxic activity, and the activity of the nervous system.

In extremity surgery, as in most surgery, the local loss of circulating fluid is the overwhelmingly important cause of surgical shock. It may be lost as blood, as plasma, or as a combination of the two. The amount of external hemorrhage from an accidental wound may not be known in case the patient has been transported before being seen by the surgeon, and too frequently, in both accidental and operative wounds, the external hemorrhage is not adequately considered as a cause of shock. This is true to an even greater extent in the case of internal hemorrhage. Crushing injuries of the thigh, especially with fracture of the femur, are particularly apt to cause massive bleeding into the tissues, and multiple fractures of smaller bones may act likewise. When tissues are traumatized, a considerable amount of the local fluid loss is plasma which may spread for some length in the adjacent soft parts. A mixture of blood and plasma is found in the tissues of limbs of animals hammered for the production of shock, and, usually, the heavier the hammer blows the greater the proportion of blood to plasma because of the injury to the larger blood vessels. Careful inspection and measurements of the injured regions and comparison with measurements of the opposite side are important measures for the recognition clinically of the amount of local fluid loss in extremity injuries.

Plasma alone is lost when there have been burns, prolonged application of a tourniquet (four hours or more), or extensive acute thrombophlebitis of the iliac and femoral veins. In a patient with thrombophlebitis of the left common iliac vein, rapid swelling of the lower limb with symptoms of shock occurred within ten hours, and death in less than twenty-four hours. At autopsy, the left common iliac vein was found completely blocked by a fresh thrombophlebitis, and measurements showed the volume of the left limb to be approximately 4000 c.c. larger than that of the right. The swelling was due principally to plasma loss into the tissues, and was the main cause of death in shock. Red-cell counts, hemoglobin, hematocrit, and plasma-protein determinations are helpful in the diagnosis of shock due to local loss of circulating fluid, but the findings are variable due in some measure to the extent to which blood, plasma, or a mixture of the two is lost. In shock due to hemorrhage, there is a greater tendency to hemodilution, although there may be hemoconcentration, especially in the severer stages. In shock due mainly to plasma loss, as in burns, there is regularly hemoconcentration. In shock due to both blood and plasma loss in considerable amounts, as in crushing wounds or in experimental shock from limb hammering, there may be little change in composition of the blood or there may be either hemodilution or concentration, according to whether there is greater loss of whole blood or of plasma.

In case of shock due to injuries, periodic rapid estimations of the circulating blood volume by use of the Evans blue-dye rapid method may be of considerable help in determining the amount of local fluid lost at the sites of injury and the amounts of blood, plasma, or both that should be administered. Since this procedure has been carried out in severe injury cases in our Clinic by Dr. Paul Schafer, it has been found that very much larger amounts of blood and plasma are lost than were formerly recognized. This has led to more

* Read at the Annual Meeting of The American Orthopaedic Association, Cleveland, Ohio, June 8, 1943.

liberal use of transfusions, and in some cases as much as 5,000 to 8,000 cubic centimeters of blood and plasma have been given in the course of a few days, utilizing the circulating-blood-volume estimations as one of the guides. In accident cases, shock and death are now being prevented more often than formerly, when smaller transfusions were given because of failure to appreciate the full significance of local loss of circulating fluid.

Toxic substances that play a rôle in the causation of shock are anaesthetic agents, bacterial toxins, and metabolic products of damaged tissues. A discussion of anaesthetics would call for a special article, but it need only be said that, when prolonged general anaesthesia is necessary in patients whose condition predisposes to shock, better results are obtained by the use of ethylene or cyclopropane than ether. Spinal anaesthesia also has a very real place in surgery of the lower extremities where shock is concerned. Severe toxicity as a result of infections located in the extremities may so predispose the patient to shock that the hazard of operative treatment is enhanced.

The theory advanced during World War I (that a toxin formed in traumatized and devitalized tissues which passed into the circulation and damaged the general capillary bed, among other tissues, producing shock in a similar manner to histamine) has been given less consideration in the past ten to fifteen years, since the importance of blood and plasma loss has been realized. Also, Roome and Wilson failed to demonstrate the presence of toxic substances in greater amounts in extracts or expressed juices of traumatized tissues than in those of untraumatized tissues. In experiments on trauma of the lower limb, it was found, by comparing weights of the two extremities, that there was enough local loss of circulating fluid in the traumatized one to explain shock and death without the assumption of a toxic factor. However, during World War II there has been a revival of interest in the toxic theory, due to observations in connection with the crush syndrome. It has been reported that when limbs have been crushed for several hours by the wreckage of buildings in bombing raids, the patients may go into shock after release, without sufficient local loss of fluid into the crushed part to fully explain the condition. Also it has been reported that plasma transfusion, in quantities that have appeared to be adequate for replacement of the amount of lost circulating fluid, has failed to relieve the shock. Blalock and others have confirmed these clinical findings in experiments on animals. The assumption is that toxins formed in the damaged tissues are the responsible factor.

The oldest theory of wound shock is that it is the result of an insult to the nervous system. The trauma may be psychic or it may be of the somatic nerves of the injured field. In either case it has been widely assumed that afferent depressor impulses pass to the vasomotor center in the medulla and act reflexly on the efferent sympathetic nerves to the blood vessels, causing a fall in blood pressure and damage to the circulation which is sufficiently severe and prolonged to result in a state of shock. There is, however, a dearth of satisfactory evidence in favor of shock production either by psychic or somatic impulses.

Psychic activity that is "shocking" in the popular sense of the word may be created by such adverse special sense impulses as the sight of a hemorrhage or of an accident, or by pain impulses. Depressor impulses from the cerebrum to the vasomotor center and heart cause a fall in blood pressure, which, when marked, results in fainting. However, the reaction is short-lived, as the blood pressure rises soon after the unconscious patient assumes the recumbent position, the faint rapidly passes off, and a prolonged state of low blood pressure with damage to the tissues does not come about.

Direct stimulation of isolated somatic nerves of the limbs by pinching, crushing, cutting, or the application of an electric current of varying strengths and frequencies fails to bring about anything remotely resembling a state of shock either in animals or in man. Clinical experience shows that an operation on an extremity may be very long and very extensive without the production of shock, if care is exercised to see that there is little or no loss of blood, or if blood loss is treated simultaneously by adequate blood transfusion.

In order to test the theory that shock may be produced by the action of vasodepressor

nerves, a series of experiments have been conducted[2] in which the aortic depressor and carotid sinus nerves, which have been evolved especially for lowering the blood pressure in the procedure of pressure modulation, and which carry only depressor impulses, have been electrically stimulated in rabbits and dogs respectively. They would never be injured and cause shock in man, but the results of their stimulation would serve as an index of the effect of vasodepression produced by stimulation of any afferent depressor nerve. By these experiments it has been possible to lower the blood pressure to shock levels where it may be maintained for long periods of time. It has been surprising to find that this reflex physiological lowering of blood pressure may be tolerated for several hours before there is serious damage to the circulation; also, that if the stimulation is stopped, the blood pressure may quickly return to the prestimulation level and the animal live on much as in a control experiment. Beyond a certain point, the circulation begins to fail and the animal passes into a state of shock which terminates fatally. The length of time required to bring on circulatory damage and shock by stimulation of these nerves is so very much longer than the length of time that the blood pressure is observed clinically to be held down reflexly by adverse cerebral activity (or by manipulation within the abdomen) that it greatly strengthens the clinical impression that shock is probably never created primarily by the action of afferent depressor impulses on the vasomotor center in the medulla. Certainly this is the case for afferent vasodepressor impulses going from a wound of an extremity directly to the medulla, as judged by the ineffectiveness of direct stimulation of such nerves.

Pain, fright, and special sense impulses like the sight of blood or of an accidental wound, by reflexly lowering blood pressure through action of the vasodepressor impulses on the medulla, set up in the brain, would never act for as long a time as is required in these experiments to produce shock by stimulation of the aortic depressor nerve. The experiments greatly strengthen the clinical impression that primarily shock is probably never caused entirely by the action of afferent depressor impulses on the vasomotor center in the medulla. When animals were first bled to shock levels and the nerves were then stimulated, the blood pressure was carried to still lower levels, and the period of survival was thereby shortened somewhat. This would lend support to the theory that vasodepressor impulses may act as a secondary or contributing factor when other shock-producing factors, as hemorrhage, have initiated shock.

The gradual realization in recent years of the overwhelming importance of blood and plasma loss as a cause of shock in extremity surgery has led to improved preventive methods, and to more extensive use of blood and plasma transfusions in both the prevention and the treatment of shock. This has resulted in a steady and marked improvement in surgical results.

Local loss of circulating fluid is prevented by the appropriate use of the tourniquet and of hemostatic forceps, and by the application of permanent pressure dressings and bandages. Use of the tourniquet for periods varying from three to three and one-half hours may not be sufficient to cause gangrene of the limb, but there may be enough tissue damage to result in plasma loss in amounts sufficient to be a factor in shock production. This is a possible complication that should not be lost sight of. When the tourniquet has been on sufficiently long to produce gangrene, prompt amputation will prevent the local loss of fluid which would otherwise ensue and lead to shock.

Long operations on the upper extremity are being performed under constriction, with the prevention of blood loss by the temporary release of the blood pressure cuff at intervals, to maintain the vitality of the extremity. Extensive operations on the thigh, in which the incision must extend into its upper portion, may be performed, to great advantage, from the standpoint of shock prevention, by the use of constriction applied by the Wyeth technique. The entire extremity is prepared aseptically and draped. A Martin bandage is then applied spirally from the foot to the upper thigh. A Steinmann pin is passed through the soft parts above the greater trochanter, and a heavy rubber tube encircling the hip is

tied above it. Extensive osteomyelitis and tumors of the femur that permit of local excision may be best operated upon in this way. If all identifiable vessels are clamped and tied during the operation, there may be little blood loss on removal of the constrictor.* In case of extensive oozing from bone and soft parts, following operation for osteomyelitis, the wound may be packed with gauze and the skin tightly sutured for twenty-four hours, at which time the stitches are removed.

We owe the use of compression dressings and bandages for the prevention of the local loss of circulatory fluid mainly to Dr. Sumner Koch, who in recent years has obtained striking results in extremity burns by their application following careful cleansing with soap and water. Swelling is reduced to a minimum, and plasma loss, both into the tissues and to the outside, is greatly reduced. Similar use of dressings and elastic bandages produces similar results in cases of wounds that otherwise would lead to extensive swelling.

It becomes more and more evident that the problem of shock in extremity surgery is mainly the problem of local loss of fluid from the circulation, and that its solution is by the prevention of such fluid loss or by transfusions of blood and plasma, if shock is threatened or has already been established.

REFERENCES

1. Blalock, Alfred: Principles of Surgical Care: Shock and Other Problems. St. Louis, C. V. Mosby Co., 1940.
2. Phemister, D. B.: Rôle of the Nervous System in Shock. Ann. Surg., CXVIII, 256, 1943.
3. Roome, N. W., and Wilson, Harwell: Experimental Shock. The Effects of Extracts from Traumatized Limbs on the Blood Pressure. Arch. Surg., XXXI, 361, 1935.

DISCUSSION

Dr. John Fulton, New Haven, Connecticut: I would agree most heartily with what has been stated concerning the influence of local fluid loss and its importance in bringing about the phenomenon of circulatory collapse that we refer to as shock. I believe, however, that the nervous factor in shock is one that has not yet been thoroughly analyzed, and that it is a factor of vital importance. Within the past year Dr. Swingle, of Princeton, and Dr. Winternitz, working from another angle in New Haven, have shown that a traumatic insult, sufficient to kill the majority of animals if untreated, or untouched from the point of view of the nervous system, will fail to kill them if the extremity in question is denervated. Swingle has further established that bilateral section of the splanchnic nerves will give a marked degree of protection from localized trauma.

Dr. A. Wickler, in my laboratory, has been interested in the subject from a more practical standpoint, and has been attempting to find agents that might suppress the nervous factor. We chose morphine as a possible agent in this connection. Morphine has been for some time a favorite drug with military surgeons, and it was found, among other things, that the effects of morphine on the nervous system had been little studied, particularly with regard to its site of action. Dr. Wickler found, on administering morphine, that the knee jerks and certain other reflexes of the spinal arc were promptly increased, both in monkeys and in cats, while pain reflexes disappeared entirely. Morphine evidently acts upon the sensory fibers belonging to the B and C groups, and has little action on the A fibers that mediate the knee jerk. This phase of the analysis was carried out by Dr. David Lloyd of the Rockefeller Institute. He found that morphine tended to abolish slow fiber action potentials in the spinal cord, while leaving the more rapid components. We hope in the near future to carry out a further analysis on the effect of morphine, because it is likely that its primary seat of action is the spinal cord, that it is selective, and that it abolishes pain reflexes that play such a large part in bringing about the circulatory collapse in which all are deeply interested.

Major General Norman T. Kirk, Surgeon General, United States Army: I would like to know what kills the soldier whose death cannot be accounted for by hemorrhage, if it is not shock. Death occurs following blast, both in air and water, and before there is loss of fluid into the tissue. We are told by the Navy that if two men are at a given distance from a depth charge, one swimming on his back and the other on his abdomen, the former will survive and the latter will be killed. The effect of blast in water is four times greater than in the air, and its action likewise reaches four times the distance from the explosion of the bomb.

It was learned in World War I in the British Army that by using the Thomas Splint to immobilize frac-

* At the meeting, lantern slides were shown of a chondrosarcoma in which, with this type of constriction, six inches of the shaft of the femur, with some of the surrounding muscle, was excised and the defect filled immediately with two large grafts, one taken from either tibia. Despite the enormous extent of the three incisions (twenty-eight inches), the extensive traumatization, and the devitalization of the tissues (large grafts), there was no shock because there was no blood loss. Convalescence was uneventful and excellent function resulted.

tures transported from what corresponded to our Clearing Station to our Evacuation Hospital, the death rate was reduced from 50 per cent. to 15 per cent., and that immobilization lessened shock and pain. Whether it made a great difference in the amount of plasma lost in tissues, I am unable to state.

It would seem that this type of death is from a form of shock which cannot be explained by fluid loss. I am satisfied, however, that a great deal of the shock that does occur is the result of fluid loss into the tissues.

Dr. Steele F. Stewart, Honolulu, Hawaii: Had I realized before I left Honolulu that this subject was coming up at this meeting, I would have gotten some data about shock from Lieutenant Commander Louis Gunther of the Aiea Naval Hospital. Some of his work will appear shortly in the Naval Bulletin and elsewhere. He has definitely shown, in both animals and man, that prior to the onset of shock there is a drop in muscle tonus or pressure, and restoration of muscle tonus or pressure restores a peripheral circulation which has failed. In all cases which he was able to follow, the drop in muscle tonus preceded the onset of shock by a considerable period of time. The application of the Thomas splint, as mentioned by General Kirk, tends to prevent shock; the same effect can be obtained in about ten minutes by the use of large doses of coramine. Also the giving of plasma gives a much more dramatic rise in the muscle pressure, but requires from fifty to ninety minutes for its complete action. I think that perhaps this agrees with Dr. Fulton's statement about the effect of morphine on the A fibers, which would suggest that, if on the operating table we could have the patients' feet supported at right angles, as by the Kenny footboard, the maintenance of muscle tonus would tend to prevent shock.

In regard to blast injuries, I would call your attention to the fact that it has been pretty definitely shown, by Lieutenant Morris T. Friedel, that the individual, who has on a life preserver of the jacket or "Mae West" type which comes down as low as the edge of the ribs, does not show any pathological changes in the chest, and that the extension of the jacket across the abdomen would tend to prevent abdominal injuries.

Dr. J. E. M. Thomson, Lincoln, Nebraska (closing): I enjoyed Dr. Phemister's paper and the scholarly discussion of Dr. Fulton in connection with the various works on general shock.

We have not exhausted our study of the phenomena which I have called "local shock". The interesting changes that take place in the tissues, the nerves, and the blood vessels which pass through the region of injury will be further explored. In several of these experimental animals, we have found something similar to Volkmann's ischaemic paralysis developing during this experiment. There may be a similarity to Zodack's syndrome. Clinical work on immersion-foot has demonstrated that blocking the sympathetics has a favorable influence on restoration of circulation. Perhaps novocain is not the drug to use. There are so many angles to this "local shock" to be solved that one could investigate any number and not solve them all. However, we have gone far enough to justify us in using novocain block clinically in certain cases of severe injuries manifesting pronounced evidence of local circulatory embarrassment and incompetence of circulation in the extremity.

Dr. Dallas B. Phemister, Chicago, Illinois (closing): Dr. Fulton believes that stimulation of the B and C fibers of the injured field may bring about shock. However, in cases of direct stimulation of a somatic nerve to an extremity by crushing or by the application of an electric current in anaesthetized animals, these fibers as well as all other fibers are stimulated, and shock is not brought on. Consequently, it is difficult to understand why stimulation of the B and C fibers alone would produce a different result.

With reference to spinal-cord injuries mentioned by Dr. Stewart, Swingle has reported that, by bilateral partial section of the cord in the anterolateral regions, shock may be produced in dogs. This diminishes muscle tone, and it is possible that blood accumulation in the flaccid hind limbs might affect the blood pressure. If so, it is surprising that complete section of the cord at various levels as high as the sixth cervical vertebra does not produce shock.

As to blasts producing rapid death, the mechanism is not that of shock, which is a peripheral circulatory failure. It may be the result of reflex failure of the heart or of respiration through concussion of the medullary centers. During dissection of the carotid sinuses for direct electrical stimulation in some shock experiments, it was not rare for sudden death to be produced reflexly, but it would be inappropriate to speak of the condition as surgical shock.

News Notes

The Twelfth Annual Convention of **The American Academy of Orthopaedic Surgeons** will be held at the Palmer House, Chicago, January 23, 24, 25, and 26, 1944, under the presidency of Dr. M. N. Smith-Petersen. Registration will begin Saturday morning, January 22, 1944, and Scientific Exhibits, Technical Exhibits, and Scientific Motion Pictures will be open Saturday afternoon. The various committee meetings will be held Saturday afternoon and Sunday morning. All day Sunday will be given over to Instructional Courses, arranged by Dr. J. E. M. Thomson, and on Monday morning the Courses on Reconstruction Surgery will be offered to the entire assembly.

The clinical program of the Clinical Orthopaedic Society will be presented Monday afternoon, followed by the Executive Session of the Society, Dr. M. O. Henry, President, presiding. On Monday evening, January 24, 1944, there will be an informal dinner followed by a panel discussion.

The annual dinner of The Academy will be held on Tuesday evening. All meetings will be informal. Ladies are not invited during wartime. Executive Sessions of The Academy will be held at noon Tuesday and Wednesday, and the following scientific program for Tuesday and Wednesday has been arranged by Dr. W. P. Blount:

TUESDAY, JANUARY 25

Morning Session

Acute Osteomyelitis with Staphylococcus Septicaemia.
Lenox D. Baker, M.D., Durham, North Carolina.

Penicillin in the Treatment of Septic Gunshot Wounds.
Captain José M. Ferrer, M.C., U. S. A.

Non-Union of the Tibia.
Arthur G. Davis, M.D., Erie, Pennsylvania.

Amputation of the Leg with Induced Synostosis of the Distal Ends of the Tibia and Fibula.
C. Glenn Barber, M.D., Cleveland, Ohio.

Amputation for Chronic Osteomyelitis.
J. Albert Key, M.D., St. Louis, Missouri.

Afternoon Session

Intravenous Morphine.
Major Hugh Smith, M.C., U. S. A., Maxwell Field, Alabama.

The Diagnostic Value of Synovial-Fluid Examinations.
Colonel Walter Bauer, M.C., U. S. A., Dallas, Texas.

The Detailed Operative Technique for Open Reduction and Internal Fixation of Long-Bone Fractures.
Clay Ray Murray, M.D., New York, N. Y.

The Use of the Stader Apparatus in Fresh Fractures.
Captain C. M. Shaar, M.C., U. S. N.

The Application of Haynes' Skeletal Fracture Apparatus to Special Orthopaedic Problems.
Robert W. Johnson, Jr., M.D., Baltimore, Maryland.

Half-Pin Fixation of Fresh and Old Fractures.
Commander Robert Mazet, Jr., M.C., U. S. N. R., Oakland, California.

WEDNESDAY, JANUARY 26

Morning Session

Medical Audio-Visual Education in the Navy.
Commander J. S. Barr, M.C., U. S. N. R.

Injuries to the Ligaments of the Knee Joints.
LeRoy C. Abbott, M.D., San Francisco, California.
John B. deC. M. Saunders, F.R.C.S., San Francisco, California.
Frederic C. Bost, M.D., San Francisco, California.
Carl E. Anderson, M.D., San Francisco, California.

Subtibial Collateral Ligament Bursitis: A Report of Cases.
Allen F. Voshell, M.D., Baltimore, Maryland.

Quadricepsplasty to Improve Knee Function.
Lieutenant Colonel T. C. Thompson, M.C., U. S. A.

Pedicle Grafts to Deep Skin Defects of the Foot and Ankle.
Ralph K. Ghormley, M.D., Rochester, Minnesota.

Immediate Application of Free Full-Thickness Graft for Traumatic Amputation of the Finger
H. Relton McCarroll, M.D., St. Louis, Missouri.

Afternoon Session

Orthopaedic Surgery in South America.
Oscar L. Miller, M.D., Charlotte, North Carolina.
The Effect of Poliomyelitis on the Nerve Endings in Skeletal Muscle.
Eben Carey, M.D., Milwaukee, Wisconsin.
Calcification of the Supraspinatus Tendon.
M. Beckett Howorth, M.D., New York, N. Y.
A Study of the Pathological Findings in Periarthritis of the Shoulder.
Julius Neviaser, M.D., Washington, D. C.
Fractures from the Pacific Combat Area.
Lieutenant Commander M.C. Mensor, M. C., U. S. N. R.
Lieutenant F. H. Smith, M.C., U. S. N. R.
Analysis of Healing and Healing Time in 250 Cases of Fractures in the Shafts of the Tibia and Femur.
Robert V. Funsten, M.D., Charlottesville, Virginia.

The "keynote" of the 1944 Instructional Courses will be "Reconstruction Surgery of the Extremities", and to this general subject have been added courses on "Fractures" and "Lame Backs". The Instructional Section will begin promptly at 9:00 o'clock Sunday morning, January 23, and will continue through Monday, January 24. Also, a motion-picture program will be presented Saturday afternoon, January 22, for those who arrive early.

The subjects to be developed in the following courses are particularly pertinent at this time, and will be discussed by eminent teachers and authorities.

COURSE I. NON-UNIONS

Fundamentals, Necessities, and Methods for Bone-Graft Surgery.	Paul B. Magnuson, M.D.
The Use of the Iliac Graft.	LeRoy C. Abbot M.D.
Barrel-Stave Graft.	Paul Steele, M.D.
Onlay, Inlay, and Peg Graft Methods.	Frank D. Dickson, M.D.
Dual and Osteoperiosteal Grafts.	James S. Speed, M.D.

COURSE II. BONE DEFECTS

Bridging Large Defects.	J. Albert Key, M.D.
Bridging Large Defects.	Paul Steele, M.D.
Bone Lengthening and Shortening.	LeRoy C. Abbott, M.D.
Overlapping Procedure for Shortening.	J. Warren White, M.D.

COURSE III. MALUNIONS

Dislocations and Fractures of the Ends of the Clavicle.	E. Bishop Mumford, M.D.
Malunion of Fractures about the Wrist and Elbow.	James S. Speed, M.D.
Reconstruction of Malunited Fractures about the Ankle.	Harold B. Boyd, M.D.
Dysfunction of the Knee Associated with Fractures of the Tibial Plateau.	Robert D. Schrock, M.D., and Herman F. Johnson, M.D.
Malunion of Fractures about the Hip.	Herman C. Schumm, M.D.
Treatment of Malunion of Fractures of the Femur.	Edward L. Compere, M.D.
Maintenance of Position.	Captain John J. Flanagan.

COURSE IV. RECONSTRUCTION OF THE FOOT

Functional Anatomy of the Foot and Ankle.	Dudley J. Morton, M.D.
Static Deformities of the Foot.	Robert V. Funsten, M.D.
Trophic Lesions of the Foot.	Commander James C. White.
Fibrotic Lesions of the Foot.	Commander James C. White.
Extensive Tissue Loss of the Foot.	Ralph K. Ghormley, M.D.
Disorganization of the Foot by Reason of Bone Loss or Joint Disintegration. (*A.*)	Lieutenant Colonel Francis M. McKeever.
Disorganization of the Foot by Reason of Bone Loss or Joint Disintegration. (*B.*)	J. Warren White, M.D.
Fractures of the Tarsal Bones,—Os Calcis.	Harold R. Conn, M.D.
Fractures of the Tarsal Bones,—Astragalus.	Oscar L. Miller, M.D.
Irreparable Nerve Lesions Producing Deformity.	H. Relton McCarroll, M.D.

COURSE V. RECONSTRUCTION OF THE HAND

Plastic Repair of the Burned Hand.	Lieutenant Commander Paul W. Greeley.
Plastic Considerations of Burns, Scars, and Contractures of the Hands.	Captain Walter B. Macomber.
Plastic Repair of Surface Defects of the Upper Extremities.	Lieutenant Commander C. R. Straatsma.
Repair of Surface Defects of the Hands by Pedicle Grafts.	Major William S. Kiskadden.
Skin and Soft-Tissue Replacement of War Wounds of the Hand and Forearm.	Captain Preston C. Iverson.
Posttraumatic Trophic Conditions of the Hand.	Lieutenant Commander W. K. Livingston.
Composite Problems in Wounds of the Hands and Forearms.	Commander Robert Mazet, Jr.
Amputations, Prostheses, and Defects of the Hands.	Major L. D. Howard, Jr.
Review of Hand Cases Evacuated from the South Pacific.	Captain George T. Aitken.
Adaptation of Structure to Disturbed Function following Deep Burns.	Lieutenant Commander S. S. Hudack.
Comments and Conclusions.	Sterling Bunnell, M.D.

COURSE VI. SKIN PLASTICS

Principles of Skin Grafts and Flaps.	Lieutenant Colonel James B. Brown.
How to Use the Dermatome.	Earl C. Padgett, M.D.
Possibilities of Gluing Skin Grafts in Place.	Machteld Sano, M.D.
Treatment of Fifty-Five Burns Received at One Time.	Captain H. L. D. Kirkham.
Problems of Surface Restoration in the R. C. A. F.	Wing Commander Alfred W. Farmer.
Repair of Surface Defects of the Extremities.	Lieutenant Colonel George K. Lewis.
Illustrative Patients Requiring Plastic and Orthopaedic Surgery Returned from Combat Zone.	Major William S. Kiskadden.
Problems of Surface Restoration in the Navy.	Lieutenant Commander Paul W. Greeley.

COURSE VII. NERVE DEFECTS

Diagnosis.	Claude C. Coleman, M.D.
Surgical Technique.	Lieutenant Colonel Roy G. Spurling.
After-Care and Rehabilitation.	Captain W. M. Craig.

COURSE VIII. MUSCLE TRANSPLANTS

Indications for Transplantation of the Trapezius.	Leo Mayer, M.D.
Indications for the Ober Operation.	Frank R. Ober, M.D.
Indications for the Tendon Shift at the Elbow.	Arthur Steindler, M.D.
Tendon and Muscle Transplantation for Inoperable Paralysis of the Radial Nerve.	Leo Mayer, M.D.
Reconstruction of the Opposing Action of the Thumb.	Sterling Bunnell, M.D.
Reconstruction of the Gliding Mechanism of the Fingers and of the Destroyed Flexor Tendons.	Leo Mayer, M.D.

COURSE IX. ARTHRODESIS *versus* ARTHROPLASTY

Arthrodesis of the Wrist.	M. N. Smith-Petersen, M.D.
Arthrodesis of the Hip.	Melvin S. Henderson, M.D.
Arthrodesis of the Shoulder.	Arthur Steindler, M.D.
Arthrodesis of the Foot and Ankle.	Major Vernon L. Hart.
Arthrodesis of the Knee.	Major Joseph E. Milgram.
Arthroplasty of the Elbow.	Edwin Ryerson, M.D.
Arthroplasty of the Hip.	James S. Speed, M.D.
Arthroplasty of the Knee.	Fred W. Hark, M.D.

COURSE X. RECONSTRUCTION OF THE SHOULDER JOINT

Muscle and Tendon Defects at the Shoulder and Their Repair.	Harrison McLaughlin, M.D.
Fractures of the Shoulder Joint.	Otto J. Hermann, M.D.
Injuries to the Nerves about the Shoulder.	Bronson S. Ray, M.D.
Indications and Technique for Arthrodesis.	J. Albert Key, M.D.
Technique and Indication for Acromioplasty.	M. N. Smith-Petersen, M.D.
Colored Film of Lesions of the Shoulder Joint.	David M. Bosworth, M.D.

COURSE XI. RECONSTRUCTION OF THE KNEE JOINT

Introduction.	Colonel Leonard T. Peterson.
Knee-Joint Surgery in the Army.	Lieutenant Colonel Mather Cleveland.
Technique for Meniscectomy.	David M. Bosworth, M.D.
Fractures Involving the Knee Joint.	Major Robert P. Kelly, Jr.
Plastic Surgery on the Quadriceps.	Lieutenant Colonel T. Campbell Thompson.

Cruciate and Lateral Ligaments.	Arthur Krida, M.D.
Bursae and Capsule.	Philip D. Wilson, M.D.
Questions and Discussion.	Colonel Leonard T. Peterson.

COURSE XII. LAME BACK

Examination and Interpretation of Symptoms.	M. N. Smith-Petersen, M.D.
Roentgenology and Myelography.	Lieutenant Colonel Joseph C. Bell.
Discussion.	Theodore Willis, M.D.
Rehabilitation of Patients Disabled by Backache.	Emil D. W. Hauser, M.D.
Backaches Observed in Military Service.	Major Vernon P. Thompson.
Indications for Operative Treatment, and Choice of Operations in the Disc Syndrome.	G. E. Haggart, M.D.

COURSE XIII. FRACTURES

Fracture Healing.	Granville A. Bennett, M.D.
Fractures that Require Open Reduction.	Colonel Robert H. Kennedy.
Skeletal Traction.	Lieutenant Colonel T. Campbell Thompson.
Compound Fractures.	Guy A. Caldwell, M.D.
Postoperative External Fixation of Fractures	Major Harry D. Morris.
Fixation for Transportation (Motion Picture).	Colonel Rex L. Diveley.

COURSE XIV. TIMING AND SEQUENCE OF APPROACH TO RECONSTRUCTION OF COMPOUND INJURIES

Penicillin and Sulfonamides.	Wallace E. Herrell, M.D.
Clinical Experience in the Use of Penicillin.	Major George K. Carpenter.
Clinical Experience in the Use of Sulfonamides and Bacteriostatics.	Guy A. Caldwell, M.D.
Skin-Plastic Approach to Battle Casualties. Preparation for Future Approach.	Sumner L. Koch, M.D.
Peripheral-Nerve Surgery.	Byron P. Stookey, M.D.

COURSE XV. REMEDIES FOR NON-UNIONS, LARGE BONE DEFECTS, AND MUSCLE TRANSPLANTS

Treatment of Non-Unions and Bone Defects.	Melvin S. Henderson, M.D.
Dual Graft.	Harold B. Boyd, M.D.
Does Open Operation Delay Union?	Clay Ray Murray, M.D.
Problem of Bone Defects Resulting from War Casualties.	Commander Harry B. Macey.
The Rôle of Muscle Transplants in Bone Defects.	Oscar L. Miller, M.D.

PANEL DISCUSSION OF THREE ACTUAL WAR CASUALTY CASES, INVOLVING SOFT TISSUE (SKIN, NERVE, AND MUSCLE) AND BONE DEFECTS

Discussants: Arthur G. Davis, M.D., Chairman; Lieutenant Colonel Francis M. McKeever; Lieutenant Colonel James B. Brown; Captain W. M. Craig; Melvin S. Henderson, M.D.; Sterling Bunnell, M.D.; Lieutenant Colonel Roy G. Spurling; James S. Speed, M.D.; Wing Commander Alfred W. Farmer; Claude C. Coleman, M.D.; and Lieutenant Commander Douglas D. Toffelmier.

The **American Red Cross** will raise its 1944 War Fund during the month of March. The goal is $200,000,000.00.

The Chinese Medical Journal, the official organ of the medical profession in China, has resumed publication with headquarters in Washington, D. C. Formerly the editorial office was in Peiping, with the publication office in Shanghai. This excellent publication has been on *The Journal's* exchange list since 1933.

Announcement has been made of the acceptance, by the **National Academy of Science, National Research Council,** of a grant of $75,000.00 from the Johnson & Johnson Research Foundation. The purpose of the grant is to enable the Division of Medical Sciences of the Council to gather medical information pertaining to the War effort, and to disseminate summaries. A central office will be established in Washington, and a staff of observers abroad will work under its direction. Disease conditions will be studied in each theatre of operation. All of the material thus gathered will be compiled later as a collection of medical experience in the present War.

The Johnson & Johnson Research Foundation was established January 1, 1940, as a non-profit philanthropic organization, by Johnson & Johnson, New Brunswick, New Jersey, with the express purpose of supporting research and development of products to serve the medical profession. It has made appropriations for both fundamental and developmental investigations, and is currently sponsoring about 100 projects located in twenty-eight universities. The fields of medical interest which have largely been supported are pharmacology (including antiseptics), allergy, physiological studies in pediatrics, and human fertility.

Readers of *The Journal* will notice a change in the appearance of this issue. In order to cooperate in the conservation of paper as requested by the Government, without sacrificing the quality of the paper on which *The Journal* is printed, it has seemed wise to increase the size of the type page, using very narrow margins. While this makes the appearance of *The Journal* less attractive, it means that the same amount of material requiring 288 pages under the old format now uses only 217 pages. (This issue includes a total of 260 pages.) Thus this change will not only result in a saving of paper, but will make each issue lighter in weight. Since we are now prohibited from using cartons in mailing *The Journal*, it is hoped that this lighter issue will reach the readers in better condition than would a heavier number.—*The Editor.*

The **Center for Research and Instruction in Physical Medicine** has recently been established at the Graduate School of Medicine of the University of Pennsylvania at Philadelphia. For this Center, The National Foundation for Infantile Paralysis has made a grant of $150,000.00 for a five-year period. The objective is the establishment of a sound scientific basis for this branch of medicine.

The plans include: (1) a center for the development of physical medicine as a scientific part of the practice of medicine; (2) a training center for medical leaders and teachers in this field; and (3) a school for training technical workers under the guidance of such professional leadership.

The Departments of Anatomy, Physiology, and Pathology of the University of Pennsylvania will cooperate with The National Foundation for Infantile Paralysis in carrying out this program.

Sir William Arbuthnot Lane

The July issue of *The British Journal of Surgery* (XXXI, 1, July 1943) includes an excellent tribute entitled "In Memoriam: Sir W. Arbuthnot Lane, Bart., C.B. (1856–1943)".

Sir William Arbuthnot Lane died January 16, 1943, at the age of eighty-seven. He had been affiliated with Guy's Hospital since he was sixteen.

Sir William made many contributions to surgery. He was an advocate of the "no touch" technique in bone surgery. His surgical work was characterized by originality in conception and perfection in execution. He initiated the practice of rib resection for empyema; he was one of the first to ligate the internal jugular vein for thrombosis from middle-ear infections; and he devised an operation for repair of cleft palates. Many were the innovations introduced by him in the surgery of fractures.

His practice of removing the entire colon for chronic intestinal stasis met with much opposition and was finally found to be of no value. He was always sincere in his beliefs and thrived on criticism. He once stated that "one should be thankful for one's enemies because they kept you stimulated".

Sir William de Courcy Wheeler, M.D., F.R.C.S.I.

The Medical Press and Circular for September 29 (CCX, 210, 1943) reports the death of Sir William de Courcy Wheeler. He died in Scotland on September 11, while serving in the Royal Navy as Temporary Surgeon Rear-Admiral. He came of a family which had a medical tradition, and he did much to enhance that tradition. Sir William was educated in Dublin and Berne. He was a hard worker, a great reader, and in touch with most of the surgical literature. He believed in travel and "seeing for himself", and was a frequent visitor to America.

He was a general surgeon, but had a great liking for orthopaedic surgery, and was encouraged in this by his friend, the late Sir Robert Jones. After the last War he was head of the great orthopaedic hospital at Blackrock, near Dublin.

Many honors came to Sir William. He was a Past President of the Royal College of Surgeons of Ireland and an Honorary Fellow of the American College of Surgeons.

He served in the R.A.M.C. in the last War, and, during the present War, he was consulting surgeon to the Navy in Scotland with the rank of Temporary Surgeon Rear-Admiral.

Sir William was a prolific writer, and many surgical journals have published papers from his pen. He was an active member of the British Medical Association, and at one time was President of the Leinster Branch. He made and kept many friends, and at the time of his death was perhaps the best known of Irish surgeons.

Current Literature

THE FOOT. Norman C. Lake, M.D., M.S., D.Sc. (Lond.), F.R.C.S. (Eng.). Ed. 3. Baltimore, Williams & Wilkins Company, 1943. $5.00.

The first 100 pages of Dr. Lake's book, treating of pathological and static conditions in the foot, are devoted to the development of the foot embryologically (indicating a close relationship between man and the apes), and to a comprehensive review of its anatomy, the human gait, and a chapter on footwear. This entire section is interestingly written and is helpful to one approaching the study of the clinical aspects of foot troubles for the first time.

The subjects of abnormalities, injuries, and their treatment, and infections are well discussed. Then follows a thorough presentation of the distinctions between flat-foot in its different varieties and weak or strained feet.

In the last half of the volume, the author deals with hallux valgus and other deformities of the great toe; corns, warts, and disturbances of nail growth; metatarsalgia; trench-foot, frost-bite, and vasospastic lesions of the foot; new growth, benign and malignant; club-foot; and several other minor affections.

The subject matter is well presented, and the book will be found very useful to anyone interested in the lesions to which the foot is commonly subject.

PICTORIAL HANDBOOK OF FRACTURE TREATMENT. Edward L. Compere, M.D., F.A.C.S., and Sam W. Banks, M.D. Chicago, The Year Book Publishers, Inc., 1943. $4.25.

There is no need for apology on the occasion of the publication of this short manual on the treatment of fractures by Compere and Banks. So many treatises on the subject have appeared in the years since, under pressure from the Fracture Committees of the American College of Surgeons and the Fracture Services of most of the metropolitan hospitals, surgeons all over the country have concentrated on bettering the management of fractures, that the time has come in which a selection should be made of a few of the most practical methods from which general practitioners may select the one best suited to their particular needs and capabilities.

The authors have done this most admirably. They have laid down the fundamentals upon which healing of osseous tissue depends, and have stated the principles which govern the process of restoring and maintaining continuity to injured bone while solidification is going on in the callus.

One hundred and seventy-two line drawings and roentgenograms make very clear the techniques for reduction of various types of fracture, which have proved satisfactory in the hands of the authors.

The clarity of the text and the excellence of the press work combine to make this book a most desirable addition to any physician's working library.

SURGICAL ERRORS AND SAFEGUARDS. Max Thorek, M.D., LL.D., D.C.M., F.I.C.S. Ed. 4. With a Foreword by Sir Hugh Devine, M.S., Hon. F.R.C.S. (Eng.), and a Chapter on Legal Responsibility in Surgical Practice by Hubert Winston Smith, A.B., M.B.A., LL.B., M.D. Philadelphia, J. B. Lippincott Co., 1943. $15.00.

"This book is intended to keep alight the danger signals, so that no point wherein pitfalls lurk shall be overlooked. The primary purpose of the work is to warn and to guide, mainly the younger surgeons and those of limited experience."

The subject is discussed in connection with surgical operations in general, bad-risk patients, blood transfusions, plastic surgery, electrosurgery, head operations, neck operations, thorax and breast, abdomen, stomach, intestines, biliary system, hernias, urinary tract, gynecology, spine and spinal cord, and extremities, and other orthopaedic conditions.

The final chapter, by Hubert W. Smith, M.D., is on the legal responsibility in surgical practice.

There is much of interest for the orthopaedic surgeon in a number of chapters, but certain points are brought out of particular interest to him. Many excellent points are made about how to, and how not to, apply plaster casts. Pain experienced after a cast is applied is a warning that something is wrong and an investigation must be made. When a skin-tight cast is used, a longitudinal layer of cardboard should be used so that the cast can be split without injuring the patient. A walking cast should never be used in a case of bimalleolar, trimalleolar, or quadrimalleolar fracture of the ankle.

In internal fixation of bones, beef bone, ivory, cow's horn, and heterogeneous human bone are not satisfactory and should not be used. Silver wire is not strong, vitallium is brittle and not malleable, vanadium still corrodes, ordinary steel sets up a reaction, but stainless steel is satisfactory. It is wise to wait six to twelve months after infection has been present before using internal fixation. Short plates should not be used.

Calcified bursae are best treated by needling. All others should be excised, never incised. Saws used in cutting bone should be kept cool by dripping saline on them in order to avoid heat necrosis. All nerves cut in amputations should be pulled out one inch, injected with absolute alcohol, and the ends cut off.

Such are examples of the positive statements made, most of them sound, but others still open to argument. The book is the fourth revision of the original, and an attempt has been made to keep it up to date. The subjects are well arranged and splendidly illustrated. There is no surgeon alive who could not avoid many pitfalls by carefully reading this excellent treatise.

REHABILITATION OF THE WAR INJURED. A SYMPOSIUM. Edited by William Brown Doherty, M.D., and Dagobert D. Runes, Ph.D. New York, The Philosophical Library, Inc., 1943. $10.00.

This publication is a compilation of articles relating to rehabilitation of the war injured, the majority of which have already been published. The book is divided into sections dealing with the various regions and phases of the problem.

Neurology and Psychiatry: This section deals mainly with the treatment, sequelae, and rehabilitation of injuries of the central nervous system. There is also an article dealing with malingering and one on resocialization of the psychotic patient.

Reconstruction and Plastic Surgery: The majority of the articles under this section deal with reconstructive surgery of the face. Several articles are general in nature.

Orthopaedics: This section is rather narrow in its scope, the articles dealing mainly with amputations, leg lengthening, arthoplasty of the hip, and an article on the transplantation of a toe to replace a thumb.

Physiotherapy: The articles in this section are more or less general in nature; however, several articles are specific regarding nerve injuries.

Occupational Therapy and Vocational Guidance: The articles in this section are very timely and cover a very large and important field in the rehabilitation of the war injured.

Legal Aspects of Rehabilitation: The articles in this section cover legislation and compensation phases of the war injured.

Miscellaneous: This section deals with the vascular and neurological lesions in survivors of shipwreck. It consists of two sections: lesions due to exposure to cold, and lesions due to prolonged dehydration and malnutrition.

The editors have compiled a very comprehensive and instructive list of articles which are of timely interest and represent the foremost thought on the subjects presented by eminent authorities, and this list is worthy of consideration by all interested in this phase of medicine.

It was the feeling of the reviewer that not enough pertinent material was presented in the section devoted to orthopaedic surgery. It could be greatly enhanced by articles dealing with the management of complications following gunshot wounds of the extremities.

BACKACHE AND SCIATIC NEURITIS. BACK INJURIES, DEFORMITIES, DISEASES, DISABILITIES. WITH NOTES ON THE PELVIS, NECK, AND BRACHIAL NEURITIS. Philip Lewin, M.D., F.A.C.S. Philadelphia, Lea & Febiger, 1943. $10.00.

It is an extremely difficult task to criticize, in the broad sense of the term, such a book as Dr. Lewin has just written. Beyond question it contains an encyclopaedic amount of information, and, if his purpose were to have it used as an encyclopaedia is used, it would then only be subject to criticism on that basis.

However, the author's preface implies that it is to be employed by the general practitioner as a guide to proper diagnosis and appropriate treatment. On that score, the reviewer can conceive of no approach to the attainment of such an objective that could be of less help than this volume of nearly 700 pages. It is essentially a compilation of recent progress in the study of vertebral lesions, with statistical tables and illustrations, in the maze of which the physician groping for help would not know what lead to follow.

It is unfortunate that the author could not have presented this valuable material in a more usable form for those who are likely to use the book.

BODY POISE. Walter Truslow, M.D., F.A.C.S. Baltimore, Williams & Wilkins Co., 1943. $4.50.

This little book is the outgrowth of lectures and teaching on posture and corrective exercises. The book is divided into three parts. The first part is devoted to a discussion of the fundamental standing position. The kinesiology of a good standing position, as well as correct foot balance, is described in detail. The muscle action about the various joints is discussed fully. This is the best part of the book. Eight other fundamental positions are described as the starting point in corrective gymnastics.

Deviations from the normal standing position in relation to the feet and back are illustrated, and exercises and apparatus for their improvement are described.

The book treats the subject of body poise in a manner not wholly in accord with the standpoint of the

orthopaedic surgeon, but, in spite of this, it contains much material which should be of value to physical educators.

ORTHOPEDIC NURSING. Robert V. Funsten, M.D., and Carmelita Calderwood, R.N., A.B. St. Louis, The C. V. Mosby Company, 1943. $3.75.

This book comprises 559 pages with 181 illustrations. It is divided into twelve units. The first unit is the introduction for teacher and student and could be read by any teacher, student, or doctor with much benefit and interest. The second unit deals with the general features of orthopaedic nursing, and gives a very detailed description of the care of patients in plaster casts; the care of the orthopaedic surgical patient; orthopaedic linen and restraints, and physical therapy. The remaining units deal with special conditions, such as congenital and acquired deformities; bone disorders in children; acute and chronic inflammation of joints; fractures, dislocations, sprains, and other joint injuries; static deformities and disabilities; conditions resulting from injury or disease of the brain, spinal cord, and nerves; bone tumors; conditions of the low back; bone disorders in adolescent and adult life; and finally the orthopaedic nurse and the War. Each of these units starts with a short explanation of the clinical condition, and this is followed by a detailed description of the nursing care of each condition.

The authors say in their preface that the book is an attempt to bring together in one volume the background of medical information and nursing techniques necessary to assist the nurse in caring for the orthopaedic patient. They have emphasized the principles of nursing procedures, realizing that a good foundation in these principles is fundamental to good nursing. The book reads easily and the illustrations are good; it is one to be recommended.

THE PHYSIOLOGICAL BASIS OF MEDICAL PRACTICE. A UNIVERSITY OF TORONTO TEXT IN APPLIED PHYSIOLOGY. Charles Herbert Best, M.A., M.D., D.Sc. (Lond.), F.R.S., F.R.C.P. (Canada), and Norman Burke Taylor, M.D., F.R.S. (Canada), F.R.C.S. (Edin.), F.R.C.P. (Canada), M.R.C.S. (Eng.), L.R.C.P. (Lond.). Ed. 3. Baltimore, The Williams & Wilkins Co., 1943. $10.00.

A new edition of this book, which is used by so many in physiology and medicine, is most welcome. Physiology is an ever-changing subject, and frequent editions of such a textbook are necessary.

One of the new contributions is the section on transfusion of blood, in which the modern use of human serum and plasma and the storage of such substances in the frozen states are reported. Much has been learned about the blood substitutes which are available, and the chemistry of the blood proteins. The new work by Landsteiner and Wiener on the *Rh* factor adds another problem to the blood-grouping technique, and explains some of the transfusion reactions.

The section on blood coagulation has been changed a good deal because of a better understanding of the mechanism involved. The modern work on prothrombin is included.

Additional information is included on the nature of shock and its treatment. The correction of the abnormal physiology caused by the shock is the basis of the treatment. The control of hemoconcentration seems to be the important guide in the control of shock due to burns. The concentration of histamine developed because of the burns is considered by some to be the toxic factor.

A section has been written by Dr. A. M. Wynne on intracellular respiration, respiratory enzymes, and energy liberation, which gives a better understanding of the part played by the catalytic systems in the control of oxidation of metabolites and liberation of energy within the cell.

Important advances have been recorded in the chapter on Metabolism and Nutrition. This is due in large measure to the progress in the work on vitamins, and especially to the work on the products of vitamin-B complex.

The book is indispensable to the research worker who wishes to correlate physiology with clinical medicine.

THE HOSPITAL IN MODERN SOCIETY. Edited by Arthur C. Bachmeyer, M.D., and Gerhard Hartman, Ph.D. New York, The Commonwealth Fund, 1943. $5.00.

The editors of this "collection of readings" are well known in the Hospital field,—Dr. Bachmeyer, as a distinguished administrator in Cincinnati and Chicago and as a pioneer organizer and teacher of courses in hospital administration, and Dr. Hartman, as Secretary of the College of Hospital Administrators and now Director of Newton Hospital.

The material in the book is culled from the "literature in the hospital field and in the allied fields of medicine, public health, management and organization, law, sociology and psychology". It covers the period 1930 to 1940.

The editors state that the volume is intended for (1) the hospital administrator or his assistant, (2) department heads, and (3) the student in hospital administration.

The articles are listed under twenty-nine chapters. The headings range from Chapter 1, Historical Review, with scholarly contributions by Dr. S. S. Goldwater and Dr. N. W. Faxon, through the duties and responsibilities of Trustees, Staff, and Hospital Administrator. Then come articles on the School of Nursing, the different professional services, the Out-Patient Department, and the laboratories. The administrative departments, covering the methods of admitting and discharging patients, financial control, construction, maintenance, dietetics, laundry, and others follow. Personnel and public relations, group hospital and health insurance, and public health are all considered. Each chapter is followed by references for further reading by the student using this textbook.

It is an excellent and useful collection. Of course, it is possible to criticize any book and call attention to omissions or what the critic considers defects, just as it is easy for the armchair strategist to find fault with the conduct of the War. The three things which this writer would consider important faults are:

1. The failure to mention under organization a General Executive Committee of the Staff which usually consists of six members,—namely, the Chief Physician and one other physician, the Chief Surgeon and one other surgeon, the Chief of one of the special departments, and the Director of the hospital who is Secretary *ex officio*. The main functions of this Committee are to consider professional matters connected with care of the patients, to advise the Director or through him the Trustees, and to nominate for Staff positions. The old method of handling these functions through the whole Staff, or through committees of the Staff without the Director, is clumsy and ineffective. Most progressive hospitals have a General Executive Committee of the Staff which is one main reason for their progress.

2. The omission of the report of Rufus Rorem or that of Dr. Haven Emerson upon the Baker Memorial, a hospital for people of moderate means, a part of the Massachusetts General Hospital. It was made a sizable unit of 320 beds to reduce the per capita cost. It is operated in such a manner that the patients pay cost only, without charge for depreciation or interest on the investment. The professional fee is limited to an agreed-upon schedule. The financial status of patients admitted is carefully checked.

This demonstration of what can be done to give the best of scientific medical study and care to people of moderate means at prices they can afford to pay is considered by these experts and many others as a distinct contribution toward solving one of our economic and social problems.

3. The remaining subject which might have been mentioned is the distinct opportunity which is being belatedly recognized for the improvement of our knowledge and the better care of chronic disease by attaching to general hospitals units of considerable size devoted to the care of patients thus afflicted. The argument for this seems unanswerable, but it is a long story and out of place here.

The editors are to be congratulated upon giving to the public so useful a volume. It will be read and studied by many more than the limited groups for whom it was chiefly prepared, and will be hailed by those groups as a book of great assistance to them.

A Hundred Years of Medicine. C. D. Haagensen and Wyndham E. B. Lloyd. New York, Sheridan House, 1943. $3.75.

This historical review of medicine, having been written primarily to interest laymen, escapes the criticism that might well be levelled at it had not the authors stated their object in the preface.

The orthopaedic section, which is the only one under review here, cites the work of André, Delpech, and Bernard Heine, pioneers who wrote and taught the subject in France and Germany in 1741, 1828, and 1838, respectively. In 1817, an orthopaedic hospital was established in Great Britain.

America followed suit with clinics at Bellevue Hospital in New York and the Good Samaritan Hospital in Boston in 1861.

Ollier's studies on the growth of bone culminated, after ten years of research, in the publication of his "A Clinical and Experimental Treatise on the Regeneration of Bone and the Artificial Production of Osseous Tissue", which gave a great impetus to the development of orthopaedic knowledge.

Others who were prominent in contributing to the progress of the specialty were MacEwen, Nicoladoni, Billroth, Hibbs, Hugh Owen Thomas, and Sir Robert Jones.

The gradual assumption by orthopaedic surgeons of the care of fractures, since World War I, has resulted in the organization of fracture clinics in the large hospitals the world over, in which both general and orthopaedic surgeons have participated.

In the final paragraphs, reference is made to the efforts of the osteopaths and chiropractors to gain recognition as practitioners of medicine.

One wonders why the illustrations are placed as they are throughout the book. For example, the only illustrations in the orthopaedic section are one of a surgeon placing a ligature on an artery, and three showing the evolution of the hemostatic forcep.

The 1943 Year Book of Industrial and Orthopedic Surgery. Charles F. Painter, M.D., Editor. Chicago, The Year Book Publishers, Incorporated, 1943. $3.00.

The series of Year Books are of special value to the general practitioner who has not the time to delve

into the literature of each specialty. These books give him in a concise form the important advances in each branch of medicine during the previous year.

The real value of the Year Books, therefore, depends on the selection of the articles from the voluminous literature, and their arrangement into a useful handbook. Again the publishers are fortunate to have an editor of The Year Book of Industrial and Orthopedic Surgery, whose maturity of judgment is evident in the careful selection made of articles of orthopaedic and traumatic interest.

This year the emphasis has been on the conditions which will be met by young surgeons faced with war casualties, and by industrial surgeons in war plants where accelerated production and constantly changing personnel have increased hazards and multiplied injuries.

Other subjects which have had special attention in the field of orthopaedic surgery during the past year are also given their share of attention,—such as the use of sulfa drugs in septic compound fractures and osteomyelitis, the respective values of external and internal methods of fixation of fractures, and the newer concepts of treatment of infantile paralysis.

This compact volume will be a real help to the man who has not been able to read the current literature in this field but wishes to be up-to-date on the subject.

AN UNUSUAL CASE OF HEREDITARY FIBROUS OSTEODYSPLASIA (FRAGILITAS OSSIUM) WITH REPLACEMENT OF DENTINE BY OSTEOCEMENTUM. K. H. Thoma, M. C. Sosman, and G. A. Bennett. *American Journal of Orthodontics and Oral Surgery* (*Oral Surgery Section*), XXIX, 1, 1943.

Two unusual cases, in a mother and daughter, from a family where deafness, bone fragility, and bone deformities, especially bowing of the legs, have been characteristic, were discussed first from a roentgenographic point of view by Sosman; second, from a pathological point of view by Bennett; and third, from an oral point of view by Thoma.

Sosman stated that an exact roentgenographic diagnosis in these two patients was difficult. There were six possible diagnoses of which fragilitas ossium (osteogenesis imperfecta) was the most likely, because of the hereditary and familial characteristics in the patients.

Bennett felt that, since there appeared to be a hereditary factor in the case, and since the predominant changes in the osseous lesion consisted of accelerated but imperfect bone formation and active lacunar resorption with resultant fibrous-tissue replacement, the descriptive diagnosis of hereditary fibrous dysplasia of bone seemed justified.

Thoma stated that ten teeth were studied, all of which showed extensive resorptive processes affecting primarily the dentine, but also involving the cementum. This resorption was repaired by trabeculated osteocementum causing a complete change in the histological appearance of the tooth.

Two conclusions could be drawn from the histological study of these teeth:

1. Teeth may absorb in a way similar to bone.
2. The fact that osteoclasts appeared in the dentine that contained no enclaved bone cells should disprove the theory that they were a conglomeration of liberated bone corpuscles.—*Kurt H. Thoma, M.D., Boston, Massachusetts.*

AN APPLIANCE FOR USE IN THE CONSERVATIVE TREATMENT OF COLLUM FRACTURES OF THE MANDIBLE, IN MAINTAINING VERTICAL DIMENSION OF THE JAW AND OVERCOMING SPASM OF THE LEVATOR MUSCLES OF THE MANDIBLE. Loren W. Gruber and John Lyford, III. *American Journal of Orthodontics and Oral Surgery* (*Oral Surgery Section*), XXIX, 160, 1943.

The authors described an appliance with a jackscrew which is to be used on partially edentulous patients in order to overcome fragment overriding at the neck of the condyle or ramus. The appliance consists of an upper and a lower denture or partial denture, the jackscrew being interposed at the tuberosity. By winding it, the distance between the tuberosity and the alveolar ridge of the mandible is increased, thus bringing down the ramus and reducing the overriding of the fragments. It is an appliance which will aid in preventing deviation and occlusional disturbances in the case of condylar fractures.—*Kurt H. Thoma, M.D., Boston, Massachusetts.*

EMBRYONAL CARCINOMA PRIMARY IN THE MANDIBLE OF A CHILD WITH INVOLVEMENT OF TOOTH PULP. Charles M. Campelia and Paul E. Boyle. *American Journal of Orthodontics and Oral Surgery* (*Oral Surgery Section*), XXIX, 299, 1943.

The authors reported the pathological findings in a case of a malignant, undifferentiated, epithelial tumor, primary in the mandible of a nine-year-old child. No other tumor of the jaw with this histological appearance could be found in the literature.

The neoplasm might have originated either from the embryonic enclaved cell rests or from odontogenic epithelial remnants.

The tumor mass caused accelerated eruption and malposition of the teeth.

Involvement of the tooth pulp and evidence of a higher degree of differentiation of the tumor in the pulp were observed. The literature concerning the finding of neoplastic tissue in the pulp was reviewed.—*Kurt H. Thoma, M.D., Boston, Massachusetts.*

A New Method of Intermaxillary Fixation for Jaw Fractures in Patients Wearing Artificial Dentures. Kurt H. Thoma. *American Journal of Orthodontics and Oral Surgery (Oral Surgery Section)*, XXIX, 433, 1943.

A new method of intermaxillary fixation of jaw fractures for use in a patient with an edentulous maxilla was described in this article. By means of two incisions in the reflection of the mucosa, right and left to the pyriform opening into the nose, a hole is drilled through the thin cortical bone and a wire is looped subperiosteally over the margin of the maxilla forming the pyriform opening. These wires protrude through the intra-oral incision, extend over the patient's artificial upper denture, and are attached to an arch wire fastened to the lower teeth. Thus, the maxilla is fastened to the mandible, holding the interposed upper denture in place.—*Kurt H. Thoma, M.D., Boston, Massachusetts.*

Y-Shaped Osteotomy for Correction of Open Bite in Adults. Kurt H. Thoma. *American Journal of Orthodontics and Oral Surgery (Oral Surgery Section)*, XXIX, 465, 1943.

The various methods devised in the past for correction of open bite are reviewed.

Open bite with angulation in the occlusal plane, which may be due either to maldevelopment in the anterior part of the jaws, or to faulty reduction of a fracture in the horizontal ramus of the mandible, is best corrected within the dental arch.

A new method, termed the Y-shaped excision, for the surgical correction of open bite in adults with little or no mandibular protrusion was presented. The method is especially useful in cases with angulation of the mandible in the premolar or molar region.

This operation is an improvement on the V-shaped excision. It is performed from both an intra-oral and extra-oral approach on both sides of the jaw, and, therefore, requires more time than the former procedure. This is justified, however, because it prevents injury to the alveolar nerve and vessels, and gives a more accurate result.—*Kurt H. Thoma, M.D., Boston, Massachusetts.*

Ice Skater's Fracture: A Form of Fatigue Fracture. C. F. Ingersoll. *The American Journal of Roentgenology and Radium Therapy*, L, 469, 1943.

This type of fatigue fracture occurs characteristically in the fibula, about three inches superior to the distal end. Clinically the lesion simulates the well-known march fracture or the less publicized shoveller's fracture, in that it occurs without obvious trauma and without sufficient immediate pain to notify the patient of the injury. The lesion could be mistaken roentgenographically for primary malignant bone neoplasm or congenital syphilis.

The author presents three cases with the following identical findings: (1) Similar lesions were found at the same point in the lower fibula with the following distribution: one on the right, one on the left, and one bilateral; (2) each patient was nine years old; (3) all were boys; (4) the skates were tubular hockey skates with attached shoes; (5) none of the boys had ever skated before.

Roentgenograms showed subperiosteal new-bone formation in the distal portion of the fibula about one and one-half inches from the distal end, and extending up the shaft approximately three inches. Opposite the greatest thickness of new-bone formation, there was endosteal bone proliferation, increasing the density of the medullary cavity, but not obliterating it. Ill-defined lines of rarefaction were seen traversing the bone. One case showed a faint fracture traversing the cortex. Check-up studies made several months later showed spindle-shaped thickening of the shaft with definite narrowing of the medullary cavity.

The anatomical mechanics of ice-skater's fracture are discussed and illustrated with diagrams. The cause of this fracture apparently is muscle, tendon, and bone fatigue. The author suggests that an important pathogenic factor is the effect of repeated localized microtraumata.—*Richard C. Batt, M.D., Berlin, New Hampshire.*

The Effect of Irradiation upon the Composition and Vascularity of Growing Rat Bones. Charles L. Hinkel. *The American Journal of Roentgenology and Radium Therapy*, L, 516, 1943.

Previous papers describing the effects of roentgen radiation on growing rat bones have dealt chiefly with changes in length, in roentgenographic appearance, and in routine hemotoxylin and eosin histological preparations. Routine hemotoxylin and eosin preparations are unsatisfactory for the study of calcification, ossification, and vascular supply. Since no previous workers have used chemical studies or bone-ash figures for the

estimation of the mineral content in irradiated bone and cartilages, the author's research on this subject is a valuable contribution.

A beam of roentgen rays, two centimeters in diameter, was directed against the anteromedial aspect of the right knee of female white rats, all thirty days old. In this way, the epiphyseal cartilage zones of the distal femur and the proximal tibia, as well as several millimeters of the shaft of each of these bones was irradiated. A single dose of 950 roentgen units was given, using 130-kilovolt radiation. The animals were sacrificed at intervals of one, seven, and ten days as well as one and two months after irradiation. Using the contralateral bones as controls, a chemical analysis reveals that roentgen irradiation of bone causes an increased ash and fat content, and a decrease in the water and matrix values.

The second part of the paper demonstrates the effect of radiation on the blood vessels of growing bones. At intervals following the above irradiation technique, animals were killed and their thoracic aortas were injected with black India ink until the tail and hind quarters became homogeneously black. Subsequent bone sections clearly show the smallest metaphyseal capillaries filled with ink. These studies were corroborated by injecting four other animals with a lead acetate solution under fluoroscopic control and then recording the changes roentgenographically. The findings demonstrate a marked diminution in the number and caliber of the metaphyseal blood vessels several days after irradiation.

The author concludes that roentgen irradiation of the femora of young growing rats is followed by an almost immediate increase in bone ash and fat content, associated with a drop in matrix and water. Although the mechanism of augmented mineral deposition is not clear, it may well be related to vascular damage. In the months after irradiation of the intensity used in these experiments, there was a gradual return toward normal content of ash, matrix, and fat. Although there was considerable regeneration of blood vessels, some degree of hypovascularity persisted throughout the period of observation.—*Richard C. Batt, M.D., Berlin, New Hampshire.*

Las periartritis osificantes del codo (Ossifying Periarthritis of the Elbow). Oscar R. Maróttoli y Arnoldo E. Didier. *Anales de Cirugía*, VIII, 321, 1942.

This condition has been given various other names by other authors,—such as, "muscular osteoma", "myositis ossificans", and "posttraumatic muscular ossification". These imply that the calcium is deposited in the muscle only, which is not the case. The term ossifying periarthritis is better, because the calcification is commonly in all the soft tissues around the joint. Dislocation and supracondylar fractures are the common causes of this condition.

The authors state that mobilization accompanied by early physical therapy will prevent the ossification, which, under these circumstances, is very rare. A large hematoma favors calcification, which usually begins in the brachialis anterior muscle or its tendinous insertion. The size is quite variable. One very important etiological factor is an "ossifying diathesis". Early excessive pain and stiffness, and, later, the roentgenographic appearance are diagnostic signs.

The prognosis is always guarded. The treatment varies with the stage and extent of the involvement. At first, a cast and roentgenotherapy are used, and, later, heat and active motion. Surgical removal of the calcified mass or masses is delayed until non-operative means have been tried. Only the patients with large masses are operated upon. A series of nine cases is presented, showing various degrees of involvement.—*Captain Louis W. Breck, Camp Swift, Texas.*

The Medical Treatment of Hematogenous Osteomyelitis. D. E. Robertson. *Annals of Surgery*, CXVIII, 318, 1943.

The author believes that, if patients with acute hematogenous osteomyelitis are treated by the sulfonamides early in the disease, deaths may be prevented. He found that children frequently tolerate sulfadiazine better than sulfathiazole. Because pathogenic organisms can become drug-resistant, he advocates giving large doses while the organism is still vulnerable. It may be necessary to discontinue the drug if the patient is allergic to it, but there is no evidence to indicate that patients who do not tolerate the drug well fail to benefit from its action.

An early recognition of the true character of the disease, from the history and the general and local symptoms, is essential to adequate medication. Incisional interference is not a factor in saving lives, and may have a very deleterious effect.

Synovial Cysts of the Popliteal Space: Clinical Significance and Treatment. G. E. Haggart. *Annals of Surgery*, CXVIII, 438, 1943.

Synovial cysts of the popliteal space are due to a posterior herniation of the knee-joint capsule or to hyperplasia—"fluid distention"—of one of the adjacent bursae, most frequently the semimembranosus.

In the majority of the twenty-two cases shown at operation to be true posterior herniations of the knee-joint capsule, trauma, causing forcible hyperextension of the knee joint, seemed to be an etiological factor. A history of trauma was also obtained in 75 per cent. of the thirteen cases of hyperplastic bursa, although in no instance did these injuries result in hyperextension of the knee.

According to the treatment recommended, the cyst is removed through an incision, usually in the midline, with the patient in a prone position. A tourniquet is not employed. When a communication with the knee joint is demonstrated, every effort should be made to close the opening. Following the closure of the wound, the patient is moved to the supine position and a plaster cast is applied from the high thigh to the ankle with the knee in full extension. This cast is left on for ten days. Thirty-six hours after operation, the patient is urged to start active contraction of the quadriceps.

All patients in this series exhibit a range of motion similar to that which was present before operation. All have returned to their former occupations. With one exception, there have been no recurrences. One man, three years after operation, exhibits a fullness in the popliteal space, but in no sense a round ball-like mass such as was present before surgery.

OSTEO-MALACIA AND PREGNANCY. N. A. Purandare. *The Antiseptic*, XL, 337, 1943.

The author presents a valuable study of osteomalacia, chiefly in pregnancy, and its causal relation to hyperparathyroidism. Treatment is primarily by the ingestion of vitamin D, which is believed to inhibit the hyperactivity of the parathyroids.—*Robert M. Green, M.D., Boston, Massachusetts.*

FRACTURES OF THE FEMUR IN WAR-TIME: A REVIEW OF THIRTY-TWO CASES. B. T. Keon-Cohen. *The Australian and New Zealand Journal of Surgery*, XII, 163, 1943.

A review of thirty-two femoral fractures is presented, most of them battle casualties in the Middle East, and many compound injuries. A few were fractures of the civilian type,—that is, incurred in motorcycle or automobile accidents; but all of them presented the problems of transport from field to hospital, and later of repatriation to Australia. Many lay in the field for as long as thirty-four hours before receiving aid, and many had numerous wounds besides the femoral fracture.

The Thomas splint is still the splint of choice for initial transport, but care must be exercised to prevent gangrenous sloughs over the dorsum of the foot when windlass traction is applied. The wounds were thoroughly débrided and loosely packed with vaseline gauze. Sulfapyridine was given by mouth and was placed in the wound. At the General Hospital, the fracture was (a) reduced by manipulation; (b) treated by skeletal traction through the tibial tubercle; (c) placed in adhesive traction, or placed in a hip spica, if being prepared for repatriation. Steinmann pins were preferred, as many Kirschner wires broke, and transfixion pins seemed to cause infection.

No case of tetanus or gas gangrene was encountered, despite eighteen compound injuries and one later death from septic pyaemia. Sepsis and gravitational abscesses were infrequent. There were several cases of peroneal palsy, renal colic, and ischaemia of the foot as complications. Only one death had occurred from enemy action. Stiffness of the joints, especially of the knee and hip, were treated by physiotherapy, although the author preferred to wait until the fracture had completely healed before instituting exercises to increase motion.—*D. H. Levinthal, M.D., Chicago, Illinois.*

SCAR DISABILITIES OF WOUNDED HANDS. B. K. Rank. *The Australian and New Zealand Journal of Surgery*, XII, 191, 1943.

This paper is primarily concerned with scars of healed soft-tissue wounds of the hands, although it is recognized that most war wounds are composite, as are those from grenade injuries or perforating bullet wounds. Ten per cent. of the patients admitted for plastic surgery were referred for treatment of the hands. In the early treatment, grafts are important on small wounds to prevent scar formation, even though a larger graft may later be necessary. Immobilization should be replaced by active motion as soon as possible.

In treating a healed scar, adequate excision is necessary to prevent recurrence. A full-thickness graft is taken from a non-hairy donor site, and is sutured in place with edge-to-edge approximation. Pressure dressings, marine sponges, or dental mold may be used. The primary dressing is not disturbed for ten to fourteen days, but pressure dressings are applied for another three weeks.

Gunshot wounds usually cause more damage to the extensor tendons, with scarring on the dorsum of the hand. A flap of fascia or fat should be used to cover the extensor tendon under the graft and a drain may be used if any hematoma is expected. If an abdominal flap is used, then a split-thickness graft is used to cover the raw donor site, and to prevent infection of the graft or exposed tendons. Painful finger scars adherent to bone may also be treated by a direct flap or finger-in-pocket method.—*D. H. Levinthal, M.D., Chicago, Illinois.*

THE TREATMENT OF BONE CAVITIES. Balcombe Quick. *The Australian and New Zealand Journal of Surgery*, XIII, 3, July 1943.

The author's method, which he has used for twenty years in the treatment of chronic bone cavities, accomplishes healing by providing an epithelial covering without seeking to obliterate the bone defect. The cavity is opened sufficiently so that it may be cleaned well of debris and sequestra, and the walls are "regularized",—that is, provided with smooth tapering surfaces. The cavity is packed with "B.I.P.P." (bismuth, iodoform, paraffin paste) until healthy granulation tissue is formed, which usually occurs in two weeks, but may require longer.

An accurate mold is made of the cavity, using dental composition plastic. This mold is covered with thin Thiersch grafts which are held in position on the mold by means of freezing with ethyl chloride spray. The graft-covered mold is then inserted into the cavity, and is maintained in place by a pressure dressing until the skin grafts have taken, usually in seven days or a little longer.

The author points out that saucerization of the cavity is not necessary, and that the method is, therefore, applicable for lesions near the ends of long bones. Because the grafted surface is protected within the recesses of a remaining defect, the usual objections to the thinness of Thiersch grafts are of no concern. It is necessary, however, to keep the newly formed skin thoroughly clean and dry in order to prevent future breakdown.—*D. H. Levinthal, M.D., Chicago, Illinois.*

TRATAMIENTO QUIRÚRGICO DEL PIE CAVO (Surgical Treatment of Cavus-Foot). Valentin C. Girardi. *Boletines de la Sociedad de Cirugía de Rosario*, IX, 438, 1942.

The author reports thirty-nine cases of cavus-foot treated in the Italian Hospital of Orthopaedics and Traumatology of Buenos Aires. Each case was studied clinically, roentgenographically, and electrically before any definite treatment was decided upon.

Three patients had such slight deformities that physiotherapy was all that was necessary. Nine other patients required osteoclasia and cast. The remaining cases required surgical treatment.

Cavus-foot, according to the author, is a deformity characterized by an exaggerated plantar arch, accompanied by an accentuated convexity of the dorsal region. Surgical treatment becomes necessary when the deformity, and the extent and magnitude of osseous and articular lesions make such treatment inevitable.

Numerous methods of treatment are mentioned,—such as those of Steindler, Hibbs, Kolliker, Spitzy, Galeazzi, and Valls. The Steindler technique, which is very popular, makes use of a transverse section of all the soft elements of the sole—aponeurosis, muscles, tendons, ligaments—and the anterior part of the calcaneum, by means of lateral and internal incisions. The Galeazzi method, which is next in popularity, uses the following technique: The plantar and interosseous ligaments of the mediotarsal articulation are cut laterally. The plantar muscles and plantar aponeurosis of the lower face of the calcaneum are loosened. The Galeazzi osteoclast is used until satisfactory correction has been obtained. Galeazzi further advises immobilization in a cast, and continuous traction on the level of the forefoot. Tendinous transplants may be used as the case requires.

José Valls of the Italian Hospital of Orthopaedics and Traumatology of Buenos Aires has developed a technique of his own. It consists of a cutaneous longitudinal incision parallel to the external border of the foot and two centimeters above it in the external calcaneal region. The incision extends from the posterior third of the calcaneum up to the calcaneocuboid articulation. The plantar ligaments and the muscles are loosened from their insertion on the lower face of the calcaneum. This is followed by a longitudinal incision about twelve centimeters long on the anterior surface of the foot. The aponeurosis is opened and the tendons are separated. A dorsal transectomy is then done up to the level of the mediotarsal articulation. It includes the articular faces of the calcaneum, talus, cuboid, and navicular, when that is necessary. The surfaces which are to be brought together are sutured with catgut. The wound is closed in stages. A cast is put on the leg and foot to maintain the correction obtained surgically.—*Victor Richards, M.D., San Francisco, California.*

CONSIDERACIONES SOBRE LAS FRACTURAS LLAMADAS "PATOLÓGICAS" (Considerations Regarding So-Called Pathological Fractures). Hebert Cagnoli. *Boletines de la Sociedad de Cirugía de Rosario*, X, 3, 1943.

The author introduces some very stimulating ideas regarding the nature of pathological fractures. These ideas forced themselves upon his attention in the case of a woman of sixty-four years, whom he treated for fracture of the neck of the femur, modified by a diffuse osteolytic process.

"Fractures", says the author, "are governed by two great factors: (1) a definite coefficient of severity; (2) a coefficient of resistance."

When the ratio between the two factors is disturbed,—for example, when the resistance bears little or no relation to the intensity of the trauma, we have a pathological fracture, the pathology residing chiefly in the bone. However, fractures which are not normal in their evolution or progress may also be called pathological.

Clinically, pathological fractures may be recognized on consideration of the following factors:

1. Force of the trauma and consequences produced.
2. Examination of antecedents.
3. Clinical study.
4. Roentgenographic study.
5. Biopsy study.

Under the first of these factors, consideration should be given as to whether the intensity of the trauma could have normally produced such a fracture, or whether the fracture is due to some pathology in the fractured part.

An examination and careful review of all antecedents is exceedingly important. Cancer and other osseous tumors—benign or malignant—fibrous osteitis, syphilis, tuberculosis, tabes, poliomyelitis, Paget's, Lobstein's, von Recklinghausen's, Gaucher's, Schüller-Christian's disease, *et cetera*, may have diminished the resistance of the fractured part.

The clinical study which investigates the immediate physical condition of the patient is necessarily just as important as a study of the antecedents.

Roentgenography will shed further light on any pathological condition which may have existed at the time of the fracture. Roentgenographic examinations have shown that the lung is the starting point or focus of neoplastic cells which spread throughout the skeleton. Pulmonary foci have been discovered in 98 per cent. of osseous metastases. The method of dissemination is the blood, but the primary or secondary starting point is the lung, followed by the liver, venae cavae, and portal vein. The neoplastic effect may be such as to completely dissolve the osseous tissue.

The osseous cyst is a latent infection which progresses slowly, and, in 60 per cent. of the cases, reveals itself through a pathological fracture. The most common occurrence of the osseous cyst is in the upper extremity of the humerus, femur, and tibia. The cyst grows underneath the cartilage in the metaphysis, and may even invade the diaphysis. Generally speaking, a fracture has no influence on the healing of a cyst.

In Paget's disease, the bone is poor in calcium only during the active phase of the disease. On recovery, the bone regains its normal calcification. In syphilis, the tissue becomes predisposed to fracture. In tabes there is a peculiar rarefying osteitis, due to the fact that the nervous system exercises a great influence over bone nutrition, and trophic disturbances are marked in bone, due to lesions of the sympathetic system.

All this becomes exceedingly important in industrial cases, where the legal determination of the nature of the fracture may have a decided bearing upon the judicial decision.—*Victor Richards, M.D., San Francisco, California.*

TARSO GIBOSO (Tarsal Gibbus). A. D. Maisonnave. *Boletín de la Sociedad de Cirugía del Uruguay*, XIII, 362, 1942.

Tarsal gibbus is the name given to the prominence found on the dorsum of the foot, at the level of the metatarsocuneiform joint. The condition was apparently first described in 1930 by Silfverskiöld, under the title of "pseudo-exostosis dorsal of the metatarsocuneiform joint". A similar condition occurring at the metacarpotrapezial articulation has been called "carpal gibbus".

The treatment should be by surgical removal of the dorsal exostosis. If this is contra-indicated for any reason, heat, rest, and the avoidance of pressure by shoes are recommended.—*Henry Milch, M.D., New York, N. Y.*

TUMOR MALIGNO DE EXTREMIDAD SUPERIOR DE HÚMERO (Malignant Tumor of Upper End of the Humerus). Caubarrere y Rubio. *Boletín de la Sociedad de Cirugía del Uruguay*, XIII, 499, 1942.

The authors describe a patient, aged twenty-nine years, with a malignant chondrosarcoma of the humerus. The patient had originally been seen in 1927, when there was roentgenographic evidence of a destructive lesion at the upper end of the humeral shaft. At that time, the diagnosis of sarcoma was made and a curettage was performed. Histological study of the specimen did not justify this diagnosis. The patient was subsequently given radium therapy, and a roentgenogram made ten months later showed complete recovery.

The patient remained quite symptom-free until 1940, when he again began to complain of pain, following a trivial injury to the extremity. Roentgenograms showed only a slight decalcification in the region of the greater tuberosity, somewhat above the level of the earlier destructive process. Various therapeutic measures, including radium, were instituted, but without relief of symptoms. Roentgenograms in 1941 showed progressive decalcification, with irregular mottling of the bone. Biopsy was performed at this time, and was reported as showing an atypical malignant chondrosarcoma. Surgical intervention was suggested, but was refused. Roentgenograms made ten months later showed further osteolysis, with perforation of the cortex, soft-tissue tumor, and a pathological fracture.

Unfortunately, no photomicrograph of the biopsy material is presented. The case is interesting, because of the long period of observation, and the earlier inability to find evidence of malignancy. The ultimate appearance of a chondrosarcoma may represent a new tumor formation, or it may represent the late malignant degeneration of a benign tumor, quiescent for a period of thirteen years.—*Henry Milch, M.D., New York, N. Y.*

LUXACIÓN INTERFALÁNGICA RECIDIVANTE. Tratamiento (Recurrent Interphalangeal Dislocation. Treatment). R. García Capurro y J. C. Russi. *Boletín de la Sociedad de Cirugía del Uruguay*, XIV, 36, 1943.

The authors report the successful treatment of a recurrent dorsal dislocation at the proximal interphalangeal joint of the right little finger. The operative intervention consists in the formation of a check ligament from autogenous fascia. Three small incisions are made; the first transversely on the dorsum above the metacarpophalangeal joint, the second and third longitudinally on either side of the interphalangeal joint. A fascial graft, three millimeters wide and eighteen centimeters long, is passed beneath the extensor tendon, across the dorsum of the second phalanx, just distal to the insertion of the extensor fasciculus, and into the base of the second phalanx. From the ulnar incision, one end of the graft is passed around the side of the finger in front of the flexor tendon, and is brought out at the radial end of the transverse incision. The other end of the graft is passed through the radial incision in front of the flexor tendon, and is brought out at the ulnar end of the transverse incision. The second phalanx is now flexed to about 30 degrees, and the free ends of the graft are sutured snugly to the extensor tendon. The finger is immobilized for a period of eighteen days.—*Henry Milch, M.D., New York, N. Y.*

"STRESS" OR "FATIGUE" FRACTURES OF BONE. J. Blair Hartley. *The British Journal of Radiology*, XVI, 255, 1943.

This is a short but comprehensive review of the subject. The earliest fatigue fracture reported is "march-foot", known since 1855. Large numbers of fatigue fractures have been recorded, since the first World War, in the Scandinavian and Central European countries. In America and Great Britain, the incidence of these fractures, except for march-foot, is surprisingly low. For instance, while a single German author reported on 590 fatigue fractures from field hospitals, American and British authors reported on one or two cases in their publications.

Fatigue fractures are found seven times more frequently in the metatarsals than in the tibia, and five to six times more frequently in the tibia than in femur or fibula; they are found only occasionally in the os calcis and pelvis. (In the meantime, Hartley himself has recorded a case of fatigue fracture of a dorsal vertebra. The actual figures of the incidences of fatigue fractures in the diagram which accompanies the article have been blocked out, apparently by the Censor.)

Fatigue fractures are most common during adolescence and early manhood, but may occur in young children. They occur in apparently normal bone. There should be no evidence of systemic bone disease nor any history of violent acute trauma. Hartley believes that the condition is analogous to "fatigue" or "exhaustion" fractures of metals subjected to overstress. Fatigue fractures of bone occur chiefly when the rest period is inadequate,—hence the name. Fatigue fracture should be distinguished from pseudofracture, from pathological fracture, and from spontaneous fracture. The differences between fatigue fractures and pseudofractures (also known as Looser's *Umbauzonen* or transformation zones) are tabulated; some of the main differences are: callus forms rapidly and well in fatigue fractures, but is absent or poor in pseudofractures; fatigue fracture is single, pseudofracture is multiple and often symmetrical; fatigue fracture occurs on concave surfaces, pseudofracture on convex surfaces.

Clinically, it is characteristic of fatigue fractures that pain subsides as soon as adequate rest is obtained, but returns during weight-bearing. The clinical and roentgenographic features are illustrated by a series of typical cases.

Treatment consists of a few days' rest in mild cases; of prolonged rest in more severe cases; and of immobilization in plaster in very severe cases. Return to weight-bearing should be gradual, and it is emphasized that in soldiers assignment to "light duty" is inadequate treatment; there should be complete rest at least for a number of days.

The significance of these fractures is: (1) Missed diagnosis may lead to recurrence or to involvement of the opposite side; (2) incorrect diagnosis may lead to unnecessary surgical intervention for suspected bone infection; (3) correct treatment is simple and always leads to recovery; (4) the medicolegal aspect involves medical discharge from the Army, compensation, pension, *et cetera*.

Further examinations will have to show whether at the time of symptoms there exists a deviation from the normal biological replacement of mineral salts in the involved bone.

The paper is profusely illustrated, and includes a good bibliography; it is a concise and lucid presentation of a disease which gains in importance as the number of young adults and adolescents subjected to modern military training increases.—*Albert Oppenheimer, M.D., Laconia, New Hampshire.*

"Exhaustion" Fracture of the Spine. Preliminary Report on One Case. J. Blair Hartley. *The British Journal of Radiology*, XVI, 348, 1943.

Not much has been written about "stress", "fatigue", or "exhaustion" fracture in the literature of the English-speaking countries. This type of fracture, common in Scandinavia, Central Europe, and Japan, is relatively rare in Britain. It occurs almost exclusively in weight-bearing bones. Hartley describes such a fracture in the fifth thoracic vertebral body in a healthy boy, seventeen years of age, who for three years had been driving a coal cart and had been delivering heavy coal bags, which he carried on his back. After feeling a "throb" in the chest, he suddenly collapsed a few days later with very acute pain in the dorsal region. The physical findings were not remarkable, and roentgenograms showed compression of the otherwise perfectly normal vertebral body of the fifth thoracic vertebra. The compression increased while the patient was kept in bed before a plaster cast was made. According to Hartley, this case corresponds to "stress" fractures in peripheral bones, because of the history of chronic "overload", the absence of any other bone changes, and the progressive deterioration even after the load had been removed. The abstractor has seen a number of similar instances in patients of this age group, and is inclined to believe that Hartley's interpretation accounts for a disease which could not be explained satisfactorily in the past.—*Albert Oppenheimer, M.D., Laconia, New Hampshire.*

The Rôle of Chemotherapy in the Treatment of Haematogenous Osteomyelitis. K. C. McKeown. *The British Journal of Surgery*, XXXI, 13, July 1943.

A series of twenty-six cases treated with sulfathiazole has been compared with seventy-four cases treated by the same surgical measures without chemotherapy.

In Group 1, sulfathiazole was begun at the onset of the disease and was continued for eight days, with a second course after an interval of three weeks. Surgical treatment consisted in multiple drill holes in the metaphysis with fixation of the part in closed plaster. The duration of the disease varied from three months to seventeen months, and the function was declared to be good to excellent in all cases.

In Group 2, cases were given chemotherapy, but the surgery was minimal. It consisted in incising the periosteum. Bone changes were severe, and the function only fair in all three cases.

Patients in Group 3 were given two courses of the drug and were operated upon by guttering the bone on the sixth day. Bone damage was severe, the function fair, and the duration long.

In Group 4, the sulfathiazole was given only in small doses or not until late in the disease. Again the results were poor to fair, and the duration averaged nearly a year.

As a result of these studies the author concludes:

1. The drug should be given early in order that it may reach the site of infection before pressure and thrombosis interfere with the vascular supply of the affected metaphysis.
2. A minimum concentration of 2.5 milligrams per 100 cubic centimeters of blood must be obtained, but the optimum level is just below 6 milligrams per 100 cubic centimeters. Higher levels usually cause albuminuria, this occurring more often when the urine is alkaline. The administration of nicotinic acid permits the development of a high blood level without hepatic or renal damage.
3. The use of chemotherapy limits the extent of the surgical measures required.
4. In many cases sequestration does not occur or the amount is minimal.
5. The duration of the disease is decreased by the use of sulfathiazole.—*Ernest M. Daland, M.D., Boston, Massachusetts.*

Pathogenetical Correlations between Periarteritis Nodosa, Renal Hypertension and Rheumatic Lesions. Hans Selye and E. Irene Pentz. *The Canadian Medical Association Journal*, XLIX, 264, 1943.

From their experiments, the authors have demonstrated that positive pathogenetic correlations exist between periarteritis nodosa, renal hypertension, and rheumatic lesions. In these experiments, four groups, each consisting of eight female albino rats, were first sensitized by salt treatment. Group I received no other treatment. Groups II and IV were unilaterally nephrectomized. Groups III and IV were further given injections of desoxycorticosterone acetate. Little hypertension resulted in Groups I and II, although Group II exhibited more than Group I. A sharp rise was demonstrated in Groups III and IV, with the greatest rise in Group IV. Macroscopic examination revealed many reddish nodules along the mesenteric blood vessels, strikingly similar to those seen in periarteritis nodosa. They were present in all animals of Group IV and in one animal of Group III, but absent in Groups I and II. The pancreas showed varying degrees of oedema in most animals treated with desoxycorticosterone acetate (D.C.A.), more pronounced in Group IV than in Group III. Kidney weight was increased in the groups treated with D.C.A., as was liver weight, which was considerably above normal in Group IV. Microscopic structural changes were quite similar to those seen in periarteritis nodosa, and changes in the renal vessels were essentially those observed in typical malignant nephrosclerosis.

The authors believe that periarteritis nodosa, renal hypertension, and rheumatic fever are partly caused by an abnormal adaptive response of the adrenal cortex, and that they represent diseases of adaptation. This does not minimize the rôle played by micro-organisms. Infection may act as a stimulator of adrenal activity. Lesions produced by corticoids may be sites for the localization of microbes.

The article is well illustrated with both macroscopic and microscopic views of sections from the experimental animals.

INTERNAL DERANGEMENTS OF THE KNEE JOINT IN THE CANADIAN ARMY (OVERSEAS). D. W. MacKenzie and J. A. MacFarlane. *The Canadian Medical Association Journal*, XLIX, 472, 1943.

In the two and one-half year period prior to December 1942, internal derangements of the knee joint necessitated 463 arthrotomies in General Hospitals of the Canadian Army (Overseas). The authors have reviewed 303 of these. Field units provided 77 per cent. of the patients operated upon, and Base or Reinforcement units the balance. Symptoms in 23 per cent. of the men antedated enlistment. The average age was slightly over twenty-eight years. The total disability period averaged ten weeks.

The most common knee-joint symptoms were localized tenderness, synovial effusion, restricted range of movement, atrophy of the thigh, and thickening of the capsule. Bucket-handle tear or anterior-flap tear of the medial meniscus accounted for a large percentage of injuries found at operation. Some lesions of the medial meniscus were complicated by other abnormalities, in addition to synovial effusion. Osteochondritis was present in 14 per cent. of the patients subjected to arthrotomies. Where loose bodies were found, evidence of serious infection was present.

At operation, a small anterolateral incision was the rule, and subtotal meniscectomy was carried out. The authors do not favor radical excision of the meniscus, except in rare cases. Operation shortly after injury is frowned upon. In fact, operation is advised only after complete examination and careful consideration prove it to be necessary. The incidence of operation in the authors' cases was 22.4 per cent., and this agrees with civilian and other military records. Good results were obtained in 70 per cent. of the patients operated upon. Patients with osteochondritis responded poorly, and operation is not advised, except where sequestra seriously restrict motion within the knee joint.

About one-half of the knees were immobilized, while only pressure bandages were used on the rest.

The authors stress the importance of preoperative exercise of the quadriceps muscle and progressively strenuous exertion after the operation, in injuries of this kind, to avoid atrophy of the thigh.

ESTUDIO CRÍTICO SOBRE EL TRATAMIENTO DE LA OSTEOMELITIS HEMATÓGENA. REVISION DE 300 CASOS (Critical Study of the Treatment of Hematogenous Osteomyelitis. Review of 300 Cases). Isidoro Pascau Pérez. *Cirugía Ortopédica y Traumatología*, X, 81, 1943.

Pérez has made a critical study of 300 patients with osteomyelitis, which he has collected in Cuba. Eleven of these were cases of primary osteomyelitis in adults; 81 per cent. of the involvement was in the lower extremity. Staphylococcic infection was found in 91 per cent. Of the 300 cases, eight were hypertoxic, 129 acute, 146 chronic; nine showed sclerosing osteomyelitis of Garrè, and eight Brodie's abscess. An attempt was made to determine the hemolytic power of the staphyloccal toxin. This was followed by a study of the antitoxic titer of the blood. Treatment in the hypertoxic form of osteomyelitis was directed to raising the defenses of the body. Various types of vaccines and toxoids were used. Small transfusions, 100 to 150 cubic centimeters were also given. Immune transfusions did not show any definite change in the condition. Acute osteomyelitis, if the general condition warranted, was treated by simple incision with or without perforation at the metaphyseal zone. Later the medullary cavity was canalized. Simple drainage at the metaphysis resulted in cure in fourteen of twenty-two cases. The results of total diaphysectomy have been poor. Of five cases, pseudarthroses resulted in two. Chronic osteomyelitis was treated usually by widely opening the medullary cavity, packing with vaseline gauze, and immobilization. In the os calcis, chronic osteomyelitis was treated by resection of the diseased bone with an excellent result. Most of this study was made before the appearance of the sulfonamides. In many instances, in the patients on whom it was used, it has not eliminated surgery for evacuation of pus or removal of sequestra. In the entire group of 300 patients, 245 were cured, eight were convalescing, eight had amputations, four died, and in thirty-five the final result was not known. In four, malignant neoplasm developed in areas of chronic osteomyelitis of thirty or more years' duration, and amputation was carried out in three of these. The author was able to produce local osteomyelitis by injecting neosulfamine into young rabbits which were injected several days later with one cubic centimeter of a 1-per-cent. solution of a culture of staphylococci. Localization of infection to the skeleton was obtained by local injection of a 5-per-cent. solution of sodium morrhuate or by local trauma to the metaphysis. No results of these experiments are given.—*John G. Kuhns, M.D., Boston, Massachusetts.*

TREATMENT OF ANKLE SPRAIN. OBSERVATIONS IN MORE THAN FIVE HUNDRED CASES. Paul E. McMaster. *The Journal of the American Medical Association*, CXXII, 659, 1943.

The author analyzes over 500 cases of acute ankle sprain, of which over 200 were treated by novocain injection, over 200 by taping, and sixty-eight by elastic bandage, compresses, or no treatment. All patients were in active military service. He observed that early active use materially shortened the period of disability, and that one novocain injection of all tender areas was effective in relieving the pain and allowing immediate motion and weight-bearing. Disability was entirely absent in many, and rarely lasted over twenty-four to forty-eight hours in the others.—*Brandon Carrell, M.D., Dallas, Texas.*

RADIATION THERAPY OF ACUTE SUBDELTOID BURSITIS. Arthur A. Brewer and Oscar C. Zink. *The Journal of the American Medical Association*, CXXII, 800, 1943.

The physical factors employed for the treatment of acute subdeltoid bursitis are 200 kilovolts, 18 milliamperes, and .5 millimeters of copper plus 1 millimeter of aluminum filter. A 50-centimeter target-skin distance and a 10 by 15 centimeter portal directed toward the anterolateral aspect of the involved shoulder were used. A satisfactory response to roentgenotherapy was obtained in eleven out of fourteen cases. It is noted that, if the patient is going to have response to roentgenotherapy, there will be marked relief of pain or complete cure within forty-eight hours. If the pain is not relieved, then further roentgenotherapy is not the method of choice. Cases of long standing respond less satisfactorily than cases of acute bursitis. The length of the patient's disability should then be the chief criterion for evaluating therapy in acute subdeltoid bursitis.

For the chronic case, the authors feel that irradiation therapy may be tried. They state that frequently pain is relieved to such an extent that a fairly satisfactory shoulder will follow,—at least, as satisfactory as those treated by other methods. No new symptoms or physical findings, even from the pathological study of resected bursae, were found.—*H. H. Beckering, M.D., Dallas, Texas.*

POSTERIOR HERNIA OF THE KNEE (BAKER'S CYST, POPLITEAL CYST, SEMIMEMBRANOSUS BURSITIS, MEDIAL GASTROCNEMIUS BURSITIS AND POPLITEAL BURSITIS). Henry W. Meyerding and Robert E. VanDemark. *The Journal of the American Medical Association*, CXXII, 858, 1943.

In this study, a Baker's cyst is considered to be an epithelial-like cyst which appears in the popliteal space, and is connected with the knee joint. Fifteen popliteal cysts were explored surgically, and it was found that in all of them the cyst connected directly to the knee joint through an opening in the posterior part of the capsule.

Symptoms of posterior hernia of the knee joint are not severe, the patient usually complaining of aching and stiffness of the knee. The tumor is not particularly painful, and is not adherent to the overlying skin. The duration of symptoms varied from four months to twenty years. Laboratory findings were negative.

The most common malignant tumor to be distinguished from cysts that communicate with the knee is fibrosarcoma, which is of hard, firm consistency, is more fixed to deep tissues than a cyst, and does not transilluminate.

Conservative treatment, consisting of aspiration, rest, and bandaging, is of little or no avail in the treatment of this type of tumor. Surgical treatment is the method of choice, and is indicated whenever symptoms are present. By means of blunt and later sharp dissection, the hernial sac is exposed down to its attachment to the capsule; a purse-string suture is used to close the pedicle.

Quadriceps-setting exercises are begun on the second postoperative day. By the end of the first week, the patient is allowed up and about the room, and is walking without crutches at the end of ten days. Convalescence has been entirely uneventful.—*H. H. Beckering, M.D., Dallas, Texas.*

MULTIPLE SPONTANEOUS IDIOPATHIC SYMMETRICAL FRACTURES. MILKMAN'S SYNDROME. Louis Edeiken and Norman G. Schneeberg. *The Journal of the American Medical Association*, CXXII, 865, 1943.

Diagnostic criteria in Milkman's disease, according to Leedham-Green and Campbell Golding are listed, with modifications, as follows:

Symptoms and signs:

1. Pain across the lower part of the back,
2. Pain in the lower extremities,
3. Awkward, hesitant, "duck-waddle" gait,
4. Difficulty in arising,
5. Physical examination negative, or revealing focal tender areas.

Roentgenographic features:

1. Circular areas of calcium deficiency, translucent transverse bands, or irregular pseudofractures,
2. Usually symmetrical lesions,
3. Little or no callus formation before therapy,
4. Osseous deformities noted only late.

Nineteen cases were culled from the literature, and a similar case was added by the authors. They note that the disease appears mainly in middle-aged women, and has no distinctive laboratory findings; the etiology and pathogenesis are unknown. The syndrome may represent a variant or an atypical form of osteomalacia; and the authors believe that frequently the diagnosis of Milkman syndrome is osteomalacia. It is entirely possible that many cases have gone unrecognized, and have been classified purely as osteomalacia.

The skeletal osteopathy of this condition is characterized by pain, disturbances of gait, and roentgenographic appearance of spontaneous, multiple, symmetrical, transparent bands of pseudofractures, showing little or no callus formation, which progress to render the victim bedfast. The authors' case came to autopsy, and the only findings were increased vascularity about the osseous lesions. Several reported cures have resulted from treatment with calcium, vitamin D, arsenicals, and vitamin A.—*H. H. Beckering, M.D., Dallas, Texas.*

Refrigeration Anesthesia in Amputations. Harry E. Mock and Harry E. Mock, Jr. *The Journal of the American Medical Association*, CXXIII, 13, 1943.

The authors report seventeen amputations with the use of refrigeration anaesthesia,—eight for peripheral vascular disease, and nine for trauma. They are quite enthusiastic about its usefulness, and the cases are quite convincing. An interesting discussion of the types and methods of refrigeration can be found in the article.—*Brandon Carrell, M.D., Dallas, Texas.*

Hemangioma of Vertebra with Compression of Cord. Report of a Case Cured with Radiation Fourteen Years Ago. L. Minor Blackford. *The Journal of the American Medical Association*, CXXIII, 144, 1943.

Blackford gives a résumé of sixty-five cases of vertebral hemangioma which have been reported, and presents a case of hemangioma of the eleventh thoracic vertebra which was studied very closely and thoroughly previous to biopsy. The biopsy report suggested an osteogenic sarcoma, telangiectatic type, but certain structures in the slides could not be overlooked. These were closely packed areas of large and small vessels with poorly formed walls. There was hyperplastic endothelium, and active hyperchromic spindle cells were present in the separating tissue. Extensive deep roentgenotherapy followed, and there was a gradual improvement in the condition of the patient, with relief from neurological signs and pain. Fourteen years later, the patient was hale and hearty, and a member of the Armed Forces.—*J. H. Perry, M.D., Dallas, Texas.*

Concept of Muscle Dysfunction in Poliomyelitis. Based on Electromyographic Studies. Arthur L. Watkins, Mary A. B. Brazier, and Robert S. Schwab. *The Journal of the American Medical Association*, CXXIII, 188, 1943.

Using a Grass electro-encephalographic apparatus, electromyograms were made on patients who had had poliomyelitis. The duration of the disease varied considerably, and the electromyograms were made over a period of two years. These results were compared to normal controls, peripheral nerve injuries, polyneuritis, and muscle spasm following fractures. Their work shows that, in the acute stages of poliomyelitis, some degree of paralysis must be present, for spontaneous electrical discharges to occur in a muscle at rest, while its antagonist, which according to clinical symptoms is in spasm, shows very little, if any, spontaneous electrical discharge, thus indicating no correlation with clinical spasm. Partially paralyzed muscles are hyperirritable, and the spontaneous electrical response to passive stretching is increased. These paralyzed muscles, during recovery, show increased spontaneous electrical discharges, and, when recovery ceases, the spontaneous electrical activity disappears. Activity also is absent in completely paralyzed muscles, and is not abnormal in muscle contractures. From the spontaneous electrical discharge records, paralysis is an objective sign of disease, and not a "mental alienation". "Incoordination", a somewhat misleading term, was substantiated by the synchronous individual discharges from the protagonist and the antagonist during activity of the muscles.—*J. H. Perry, M.D., Dallas, Texas.*

Sudeck's Acute Bone Atrophy. Report of Case. F. B. Brown. *Journal of the Medical Association of Georgia*, XXXII, 242, 1943.

Theories as to the causation of Sudeck's acute bone atrophy are noted. The main symptoms are extreme pain, continued swelling following an injury, with tightening of the skin, and coldness of the affected part. Roentgenograms taken after several weeks reveal a marked atrophy of the bones. Conservative treatment should be used first, consisting of diathermy, hydrotherapy, rest in bed, and the fitting of a plaster cast after the oedema has disappeared. Forceful manipulation should never be done. In stubborn cases, periarterial sympathectomy or ganglionectomy is recommended. Amputation should never be done. The author reports a case in detail.—*Fred G. Hodgson, M.D., Atlanta, Georgia.*

EFFECT OF INFRARED HEAT ON LOCALIZED POLIOMYELITIS AND NEURITIS. John A. Toomey. *The Journal of Pediatrics*, XXII, 135, 1943.

The author states that if the anterior-horn cells are all destroyed in poliomyelitis, recovery is impossible, because nerve tissue does not regenerate. When a patient has such an extensive paralysis in the initial stages of the disease that no muscle response can be elicited, and then recovers, it is because a few anterior-horn cells were left in the involved area. To prove this fact, the author reports the results of an autopsy on a child who died of pneumonia a few weeks after complete clinical recovery from poliomyelitis. Serial sections of the cord demonstrated that most of the anterior-horn cells in the lumbar area were absent, although the patient had been able to move the muscles 100 per cent. Movement is a question of balance and not primarily of great muscle strength, so that a few fibers might be able to bring about normal movement and normal balanced locomotion.

It is essential to maintain the muscle in good condition, so that the fibers remaining will have something to move. This the author accomplished by the use of infra-red heat. He studied the effect of this treatment on the thin muscles of the face in three cases of poliomyelitis and six cases of peripheral neuropathy (neuritis). All patients had severe paralysis of the muscles supplied by the seventh nerve.

The three patients with poliomyelitis were able to use the muscle voluntarily for a few minutes after the application of heat. The period of voluntary use of the muscles increased. None of the six patients with peripheral neuropathy were able to move the muscles voluntarily immediately after the treatment.

The application of infra-red rays cannot be used as a point in differential diagnosis between the two conditions, because so many patients have only partial involvement; however, it is possible to get some idea of the prognosis.

The prognosis of a peripheral neuropathy is the prognosis of the whole patient.

If, after three weeks following the disappearance of oedema in poliomyelitis, there is no response to the application of heat, the prognosis concerning the return of function becomes extremely guarded. If a patient, with a diagnosis of poliomyelitis and complete paralysis long after the oedema would ordinarily have subsided, regains function a few months later, the diagnosis of poliomyelitis becomes questionable, and that of neuritis most probable.

LOW-BACK PAIN. James H. Young. *The Medical Journal of Australia*, II, 181, 1943.

In cases of low-back pain, the author takes the patient's history, and then proceeds to a complete diagnostic manipulative examination to determine the source of the pain. This examination is described and illustrated. He bases his conclusions on 197 patients whose main complaint was low-back pain. One quarter of these had fibrositis, acute or chronic, often traced to dental sepsis. Other frequent causes were adhesions, rupture of the sacrospinalis insertion, spondylitis, and postural backache. He describes the diagnosis and treatment of these injuries, as well as sacro-iliac and lumbosacral-joint conditions, generalized spinal arthritis, and flat-back strain.

FRACTURES ABOUT THE ANKLE. J. S. Batchelor. *The Medical Press and Circular*, CCX, 138, 1943.

Fractures about the ankle are discussed and are classified under external rotation, abduction, and adduction fractures of the first, second, or third degree, as well as marginal and compression fractures. The majority of fractures about the ankle are produced by the leverage action of the foot. In first-degree fractures, there is usually no displacement, and plaster immobilization is unnecessary. In second-degree fractures, where there is slight displacement, and in third-degree fractures, where considerable backward displacement of the astragalus may be present, immobilization in a plaster cast is routine. Where immobilization in a plaster cast is not needed, an elastoplast or viscopaste bandage is used.

Practice of toe movements and contractions of muscles of the leg are emphasized as important, if a good result is to be obtained. A plaster cast applied for a fracture of the ankle should extend from the head of the tibia to the toes. After the cast is removed, an elastoplast bandage is applied to prevent oedema.

A badly damaged ligament should be immobilized for a period of from six to eight weeks. Any ruptured ligaments must be given sufficient time to heal, as laxity of any of the main ligaments may produce troublesome instability.

SOME ASPECTS OF THE TREATMENT OF WAR WOUNDS: A RETROSPECTIVE AND PRESENT-DAY CONTEMPLATION. A. E. Mortimer Woolf. *The Medical Press and Circular*, CCX, 207, 1943.

The author compares the treatment of war wounds in the War of 1914–1918 with the methods used in the present War, and describes his own procedure. In cleaning a wound, he first uses ether and then a preparation of a .5-per-cent. solution of crystal violet, and a .5-per-cent. solution of brilliant green in rectified spirit. He stresses the importance of sufficient excision of the wound. Although the sulfonamides are a help against sepsis, the author feels that, as the powder seems to prevent granulation and healing of the

wounded surface, a means of introducing and keeping a definite concentration of sulfanilamide in the blood would make local dusting of the wound unnecessary. Plaster casts used to immobilize wounds should be removed as soon as the danger of sepsis is over, if the wound is clean and healing. In packing a wound, care should be taken to ensure proper drainage; a length of gauze is laid on the skin, centering the wound; an instrument is used to press down the center and form a funnel, and the ends are folded over into the funnel made.

Present-day restorative methods make better amputations possible and eliminate many entirely.

Wounds of the knee joint afford a very good example of the benefit of modern treatment as compared with that during the last War. Today many joints are saved with mere aspiration; if there is a wound, it may have to be excised and washed out with ether, a particularly valuable treatment in streptococcal infections of joints.

In the treatment of stiff joints and tendons, one of the most important lessons learned is the institution of purposive movements at the earliest possible moment.

Osteotomía intertrocantereana con enclavación en las pseudartrosis del cuello femoral (Intertrochanteric Osteotomy with Nailing in Non-Union of the Neck of the Femur). Juan Farill. *Medicina Revista Mexicana*, XXIII, 1, 1943.

The author of this paper is making a preliminary report of a method of controlling intertrochanteric osteotomy by means of a modified Neufeld nail. He found that in an uncontrolled osteotomy, the fragments frequently became displaced, and he sought for a method of fixation.

It will be recalled that the Neufeld nail is a V-shaped nail with a trochanteric plate attached,—all in one piece. The Neufeld nail is modified by sharply increasing the angle at the junction of the nail and plate. This part fits over the trochanter, and, then, a short distance farther down at the site of the osteotomy, the plate is bent sharply in the opposite direction; the remainder of the plate extends down the shaft of the femur.

The operative technique includes the insertion of a Steinmann pin, thus stabilizing the intertrochanteric osteotomy with the upper end of the femur and neck in a marked varus position, applying the nail, and securing the plate to the shaft with screws, and, finally, withdrawing the original stabilizing Steinmann pin. No cast is applied. The author has treated four cases by this method. The article is well illustrated with drawings. Detailed case histories are not presented, since this is a preliminary report.—*Captain Louis W. Breck, Camp Swift, Texas.*

Metastatic Neuroblastoma in Bone. S. Graham Aitken. *New Zealand Medical Journal*, XLI, 209, 1942.

The author describes a tumor of the tibia in an adolescent. The tumor presented the generally accepted features of Ewing's tumor. At autopsy, an adrenal neuroblastoma with multiple secondary deposits was found.

The confusion of ideas expressed in the literature is pointed out.

The author feels that there is a lack of agreement as to what constitutes Ewing's tumor, and that many cases, reported as such, cannot be accepted without gross and microscopic postmortem findings.—*Major H. Walden Fitzgerald, Middle East Force.*

Multiple Neuritis of the Shoulder Girdle. A Report of Nine Cases Occurring in the Second New Zealand Expeditionary Force in the Middle East. E. D. Burnhard and T. G. Fox. *New Zealand Medical Journal*, XLI, 243, 1942.

A series of cases of wasting of the shoulder-girdle muscles is described. The etiology was uncertain, and there was no great tendency toward recovery. The authors suggest a form of multiple neuritis as an explanation.

Full investigations were apparently not possible. (One feels that a similar series of cases would be seen at any orthopaedic out-patient department.)—*Major H. Walden Fitzgerald, Middle East Force.*

Amputation under Ice Anaesthesia. A. Eisdell Moore and D. W. Guthrie. *New Zealand Medical Journal*, XLII, 97, 1943.

The authors describe a series of ten cases, five in detail, of gangrene of the lower limb associated with internal degeneration, for which amputation was performed successfully, usually by the Callender technique, under ice anaesthesia, and without mortality.—*Major H. Walden Fitzgerald, Middle East Force.*

An Unusual Carpal Abnormality. Eric Hudson. *Radiography*, IX, 81, 1943.

The author reports a case of fusion of the hamate and triquetrum bilaterally, associated with partial fusion of the hamate and capitate of both wrists.—*Albert Oppenheimer, M.D., Laconia, New Hampshire.*

EVALUATION OF DISABILITY IN LOW BACK INJURIES. Forrest L. Schumacher. *Radiology*, XLI, 18, 1943.

In evaluating disability in industry for compensation purposes, trauma must be separated from disease, congenital anomalies, and neoplasms. The estimate of disability must be made both as to its degree and its state,—temporary, permanent, or progressive. Such an estimation is relatively simple in severe cases, but it becomes very difficult where only the subjective symptom of pain is present. A careful history which recreates the mechanics of the accident when correlated with the physical findings is essential to prove that an injury has occurred. Delay in seeing the patient makes this difficult, but an accurate diagnosis makes the evaluation much simpler.

In order of frequency, the following are encountered: simple muscle or ligamentous strain, chronic strain, rupture of muscle or ligament, joint involvement, and any combination of these. Sacro-iliac sprain should be doubted, without direct trauma to the part or physical contact with an object. Arthritis aggravated by injury is a diagnosis to be used with caution.

Roentgenographically the majority of these low-back injuries are negative. Congenital anomalies are reported, but rarely produce disability. Hypertrophic spurs are dismissed as the ordinary result of wear and tear. Anatomically these cannot be supposed to impinge upon spinal nerves. Fractures of these spurs do occur, and thus the underlying condition is aggravated, but the spurs progress long after the injury has healed.

The evaluation of disability depends on the loss or impairment of function. McBride's schedule, based on normal as 100 per cent. efficient, is contrasted with Kessler's, which is based on two functional conditions,—limitation of motion and impairment of strength; he uses the greatest percentage of loss for the compensable disability. McBride's system attempts to evaluate function by breaking it up into component parts,—quickness of action, coordination of movement, strength, security, and endurance.—*Edward N. Reed, M.D., Santa Monica, California.*

THE VALUE OF ROENTGEN THERAPY IN CARCINOMATOUS METASTASES TO BONE. Edward C. Koenig and Gordon J. Culver. *Radiology*, XLI, 38, 1943.

Roentgenotherapy in metastatic carcinomata to bone is justified by the relief of pain and prolongation of life. Borak classifies these lesions into two groups:

1. Those which involve bone alone, producing local bone pain through periosteal involvement;
2. Those which involve the paraskeletal lymphatics or bone in such a way as to press on nerves, producing neuritic pain. Thus skeletal changes can be present without pain, and pain can be present without skeletal changes. Routine roentgenography of the skeleton, particularly of the pelvis and lower spine in breast lesions, is indicated, even without symptoms, and therapy is indicated to all lesions when found, even when asymptomatic. Also, in a case without roentgenographic findings, where it is felt that pain is a result of skeletal changes, roentgenotherapy is indicated.

Two pathological processes are described:

1. The local destructive effect of the neoplastic cells;
2. The reaction of the inflammatory zone.

Borak's thesis, that the effect produced is only upon the non-specific inflammatory reaction and on the malignant process, is considered more likely than Leddy's additional proposition of action on nerves, since hyperaesthesia of the skin is often a result of the exposure of non-pathological tissue to roentgen rays. The immediate relief of pain is attributed to the effect of the roentgen ray on the inflammatory reaction. This may be seen in five to ten days after the first treatment. A later effect, attributable to the action on the malignant cells, is also seen.

The authors feel with Borak that bone metastases are not as radioresistant as supposed. They have noted recalcification following irradiation. The measure to be used, however, in treating the patient, is his general condition and not the roentgenographic findings. Patients in good health, even with extensive lesions, represent better risks than the cachectic patient. The effects to be expected are regression of the lesion, alleviation of pain, improvement in the general condition, and prolongation of life.—*Edward N. Reed, M. D. Santa Monica, California.*

CHONDRO-OSTEODYSTROPHY: MORQUIO'S DISEASE: CASE OBSERVED DURING PREGNANCY. Peter E. Russo. *Radiology*, XLI, 42, 1943.

The case presented is that of a colored, female dwarf, twenty-six years old. She was pregnant, with the last menses in August 1941. Roentgenograms, measurements, and laboratory findings are given. The female child was delivered by Caesarean section, and died ten hours after delivery, of massive atalectasis. No other pathological changes in the child were noted.

A brief review of the literature is included. Hirsch's pathological process, which consists of three phases, is described: a *latent* stage of normal growth up to the first year; a *florid* stage in which osseous abnormalities and growth disturbances become manifest; and a third period of *repair*. Kaufmann's classification is given:

(1) hypoplastic type, in which the proliferating cartilage fails to produce enough longitudinal rows; (2) hyperplastic type, in which the cartilage is of normal hardness but of undisciplined growth, producing epiphyses of a large fungus type on normal diaphyses; and (3) malacic type, in which the cartilage is softened. All three of these types may be found in one patient, although one of the three will predominate.

Gross findings consist of: dwarfism; a normal or enlarged head sitting on the thorax with the chin on the sternum, the patient appearing to have no neck; extremities shortened; joints large, relaxed, and hypermotile; pelvis tilted forward; and coxa vara, genu valgum, pes planus, and associated deformities. The roentgenographic findings include shortening and distortion of practically every bone; absence of ossification or delayed ossification of epiphyses and epiphysioids; non-fusion of secondary centers of ossification; widening of joint spaces, with irregular and eroded articular margins; poor visualization of bone architecture; and vertebral bodies narrowed and flattened (tongue bodies). All bones appear decalcified, and the bone changes are symmetrical; numerous areas of translucency in many bones probably are due to islands of cartilage which fail to ossify.—*Edward N. Reed, M.D., Santa Monica, California.*

VENOGRAPHY WITH FLUROOSCOPY IN VENOUS LESIONS OF THE LOWER LIMB. Albert Lesser and Louis Raider. *Radiology*, XLI, 157, 1943.

Following a brief review of the literature, the authors describe a technique for combining fluoroscopy with venography. Twenty cubic centimeters of a 35-per-cent. solution of diodrast is placed in a syringe. Half of this amount is injected over a period of ten to thirty seconds, and is watched fluoroscopically. Thus the major pathological lesion is identified and localized; and observations of venous dynamics are made. The patient can then be positioned and the venogram made with the remainder of the diodrast.

Five important features of normal venograms are described:

1. The deep tributaries of the lower leg fuse to form the venae comites of the anterior and posterior tibial arteries which then fuse to form the popliteal vein.
2. The popliteal, femoral, and iliac veins fill as a straight tube.
3. In the popliteal region, a communication which often exists between the deep and superficial veins results in a filling of the long saphenous vein.
4. At varying levels, valves are seen as fine lines converging toward the center of the vein and associated with bulges in the walls of the veins which represent the paravalvular sinuses.
5. Spasm may normally be present, but its transciency and absence of collateralization distinguishes it from a true occlusion.

The pathological changes described are limited to those of occlusion and varicosities. The occlusions are classed as superficial or deep, and complete or incomplete. Varicosities are evidenced by kinks, pooling, and diminution or absence of valves. Complete superficial occlusion may involve the entire vein or be segmental, in which case collateralization of the segment is evident. Complete deep occlusion shows absence of filling, and extensive collateralization. Incomplete occlusion shows luminal narrowing and irregularities, intimal plaques, dispersal of diodrast into clusters of tributaries, and dilatation of the valve sites.

Illustrative roentgenograms of normal venograms are given. Roentgenograms and case histories are presented to illustrate the point that the clinical picture is not always reliable, and the aid obtained from a venogram may be of valuable assistance.—*Edward N. Reed, M.D., Santa Monica, California.*

GIANT CELL TUMORS: RADIATION THERAPY AND LATE RESULTS. J. Gershon-Cohen. *Radiology*, XLI, 261, 1943.

A series of twenty-nine cases are presented, with an analysis of end results following a period of observation of from five to thirteen years. Fourteen were treated by irradiation alone, fourteen by surgery and irradiation, and one received no treatment. Two cases underwent malignant change, one six years and the other nine years after treatment. Both of these patients were treated by irradiation alone, and expired nine and six years after treatment.

The rate of healing did not seem altered by the technique of irradiation. An initial osteoclasia was observed, sometimes alarming, followed by osteoplasia and recalcification. Many times a small trabeculated giant-cell tumor of benign appearance would become an osteoclastic formless mass, breaking through its bounds into the perifocal soft tissues, only to go on later to form new boundaries, envelop itself in a thin calcified capsule, and recalcify weeks or months after treatment. Such a sequence was more apt to occur if the initial roentgen-ray doses were large.

The expansile central type of tumor represents a late appearance. The tumor originates in or close to the cortex, and extends into the medullary cavity as growth progresses. A thin, calcified membrane, possibly the periosteum, seems to limit extension into the soft tissue for a while, but later, often after fracture, trauma, or irradiation, the outer envelope will be broken and the soft tissues invaded. Rarely do the tumors extend across the epiphyseal line before its closure, but in this series two cases were observed to extend across joint spaces.

The confusion in the diagnosis of malignant giant-cell tumors as made by pathologists and roentgenologists is discussed. The author is of the impression that neither can be in possession of means to foresee the probable malignant course of a giant-cell tumor. He also feels that excessive irradiation, particularly in the period of osteoclasis, may be as harmful as inadequate surgery. The two cases, which underwent malignant change, had a total of 12,000 and 6400 roentgen units, respectively. Following irradiation, the author feels expectant treatment advisable, with the use of splints to prevent fracture. This will prove rewarding in view of the biphasic phenomenon of osteoclasia followed by osteoplasia and recalcification, as well as Herendeen's paradoxical reaction to roentgen ray. When surgery is employed, postoperative irradiation should also be used to complete the adequacy of the surgical excision. In the hands of an expert, either therapeutic course seems sufficient in itself, but, in general, a patient would seem to fare better with roentgen irradiation alone, given in close cooperation with the surgeon, who will limit his aid to the use of external supportive measures,—as splinting the part and sparing it from undue strain until recalcification has been achieved.—*Edward N. Reed, M.D., Santa Monica, California.*

FRACTURAS EXPUESTAS (Compound Fractures). Juan B. Carpanelli. *La Revista de Medicina y Ciencias Afines*, V, 6, 1943.

The author begins by explaining the difference between open, compound, and other forms of fractures. A fracture is compound when bone fragments protrude from the edge of the wound or are found on its surface. The particular types of compound fractures the author mentions are those caused by firearms, vehicular collisions, and industrial accidents.

Due to the gravity of compound fractures, emergency treatment, consisting of anti-infection therapy, reduction, and immobilization, is essential. Chronic suppuration is the most frequent local complication of compound fractures. Infection is successfully combated, if treatment is started within six to eight hours after the occurrence of the accident. In a compound fracture, the wound can be closed, if the anti-infection procedure is started within that period.

Good treatment requires that the degree of shock and the general physical condition of the patient be determined as early as possible. The anaesthetics to be used must depend on the age of the patient and the type of fracture. Foreign bodies should be removed; deep sutures should be avoided; and only vessels of heavily caliber should be ligated, torsion being used in all others. Caustic antiseptics should be avoided, and, wherever possible, use should be made of the antiseptics suggested by Albert, Key, and Frankel. These suggestions include the use of powdered sulfonamides for connective tissue, muscles, and joints, while solutions of sulfonamides are recommended for pleurae and peritoneum.

It is best to use mixtures of sulfanilamide and sulfathiazole when available. Roentgenographic control is as indispensable as are frequent and complete physical examinations, for the success of the treatment.

Good circulation is favored by appropriate position, adequate warmth, and active movement of distal segments. The fracture should be immobilized for as long a time as may be necessary to obtain the best results, consideration being given to the possible existence of soft calluses. Local immobilization serves best the healing process of wounds and fractures, but there can be no objection to accelerated general mobilization and respiratory gymnastics, which may be begun at the bedside of the patient. The patient is encouraged to get up as soon as his condition permits.—*Victor Richards, M.D., San Francisco, California.*

LUXACIÓN RECIDIVANTE DEL HOMBRE. CURACIÓN (Recurring Luxation of the Shoulder. Cure). Alberto Rodriguez Egana y José Ernesto Rodrigues. *La Revista de Medicina y Ciencias Afines*, V, 89, 1943.

The authors report an unusual case of shoulder luxation.

After giving due consideration to the merits and demerits of various treatments, such as Rocher's rib-grafting; Henderson's technique of overlapping capsulorraphy, in the Mayo manner, which forms a new anterior ligament in the articular capsule; Perthes', Clermont's, and Ehrlich's methods of musculorraphy, which use a posterior fascicule of the deltoids; tenosuspension as practiced by Joseph and Loeffler; coracoid extension as practiced by Wilmoth and Tavernier, *et cetera*, the authors finally decided to use the Ricardo Finochietto method which is fully described in the *Zeitschrift fur Chirurgie* of 1918. It consists in the introduction of an osseous graft—a piece of rib—between the coracoid apophysis and a pocket formed in the subscapularis in front of the glenoid, which gives real support to the head of the humerus. Reoperation became necessary when the first attempt failed.—*Victor Richards, M.D., San Francisco, California.*

DISLOCATION OF THE PROXIMAL END OF THE FIBULA. CASE REPORT. Samuel R. Terhune, Samuel B. Thompson, and Thomas S. Eddleman. *Southern Medical Journal*, XXXVI, 271, 1943.

This is a report of a man, aged twenty-three years, who injured his leg sliding into base in a ball game. There was severe pain just below the knee. Examination showed a bony prominence at the site of the head of the right fibula, which was felt to be displaced forward and laterally. Roentgenograms showed complete dislocation of the head of the fibula forward and laterally, and no fracture of the fibula or tibia. The area

was infiltrated with 1-per-cent novocain. The dislocation was easily reduced by thumb pressure. Reduction was sudden and accompanied by a loud snap. The head was held in place by a felt pad and adhesive. There was no tendency to redislocation. The patient was kept in bed for two weeks. At the end of four weeks there was no deformity, pain, or disability, and he returned to full military duty.—*Fred G. Hodgson, M.D., Atlanta, Georgia.*

SMITH-PETERSEN NAIL FIXATION IN HIP DISEASE. J. Warren White. *Southern Medical Journal*, XXXVI, 333, 1943.

The author describes a method of producing hip arthrodesis which simplifies the technique and aftercare. The greater trochanter, femoral neck, and acetabulum are transfixed by a long Smith-Petersen nail. It prevents the postoperative tendency to adduction-flexion deformity. It is particularly suitable for tuberculous hips without too much bone destruction. The technique is the same as for fixing a fractured neck of the femur, except that no lateral roentgenograms are necessary. If correction of the deformity cannot be done by manipulation, a subtrochanteric osteotomy is also performed.

The operation was also done for two old unsatisfactory cases of congenital dislocation of the hip and for one case of osteochondritis dissecans in an adult. The operation has been performed seven times. This is a preliminary report.—*Fred G. Hodgson, M.D., Atlanta, Georgia.*

MODIFIED BONE BLOCK FOR PARALYTIC FOOT DROP. Joseph H. Gaston. *Southern Medical Journal*, XXXVI, 336, 1943.

With a specially designed hollow chisel, one-half inch in width, a section of the os calcis, including the insertion of the tendo achillis, is cut from the upper surface of the os calcis. This exposes the posterior end of the astragalus and the subastragalar joint. With the same chisel, a small section of the posterior astragalus is removed, leaving a groove for the graft from the os calcis to fit into. The graft from the os calcis is fitted into this groove and into the os calcis groove so as to block plantar flexion of the foot. The section of the bone from the astragalus is placed just behind the graft from the os calcis. A posterior flap from the tendo achillis is turned down and is fastened into the os calcis behind the grafts. The wound is closed, and a plaster cast is applied with the foot in slight equinus and the knee slightly flexed.

This article is well illustrated and shows the details of the operation and the result.—*Fred G. Hodgson, M.D., Atlanta, Georgia.*

THE RESULT OF MENISCECTOMY (KNEE JOINT) IN SOLDIERS. A. Scott Hamilton and Howard E. Finklestein. *Southern Medical Journal*, XXXVI, 406, 1943.

This is a report of 110 patients operated upon at the Station Hospital at Camp Claiborne, Louisiana. Statistics are given concerning the branch of service, mode of injury, signs and symptoms, classification of abnormalities, and postoperative complications. Indications for operative care are discussed. Particular attention is given to rehabilitation of these patients. Of the 110 patients, ninety-six returned to full duty, and the others returned to limited duty. Only two patients, in whom psychoses developed, were discharged from the Army.—*Fred G. Hodgson, M.D., Atlanta, Georgia.*

ANATOMICAL AND MECHANICAL FEATURES OF TREATMENT OF FRACTURES OF THE HUMERUS. Walter G. Stuck. *Southern Medical Journal*, XXXVI, 543, 1943.

This is a review based on 250 fractures of the humerus treated at the Robert B. Green Hospital. The author states that fractures of the greater tuberosity with marked separation usually require operation and fixation of the fragment. Fractures of the surgical neck with abduction of the upper fragment require abduction and traction on the arm to reduce the displacement. Sometimes open reduction and fixation are required. Fracture-dislocation of the head of the humerus often requires operation. The head fragment should not be removed. Fractures of the shaft often involve the radial nerve. The function of this nerve should be tested before reduction. The "hanging-cast" treatment of fractures of the shaft is often quite satisfactory. Fractures of the lower end of the humerus, involving the condyle and the elbow joint, must be very accurately reduced and maintained. Danger of Volkmann's contracture must be guarded against. The muscle pull on fragments should be considered. Open reduction and fixation of fragments are often necessary.—*Fred G. Hodgson, M.D., Atlanta, Georgia.*

ORTHOPEDIC PROBLEMS AT A NAVAL AIR STATION. E. L. Jewett and Herschel Penn. *Southern Medical Journal*, XXXVI, 533, 1943.

Orthopaedic problems for the Naval medical officer at a Naval Air Station are chiefly concerned with accidents incident to flying mishaps or crashes. Added to these are the injuries sustained while working in repair shops, or while engaging in sports at the field.

The authors have had frequent occasion to treat injuries to the semilunar cartilages of the knee. One day after the removal of the ruptured cartilage, active contraction of the quadriceps group of muscles is started. Men are returned to active duty four to six weeks after operation, with the power of the quadriceps muscle and the use of the knee joint at least 75 per cent. of normal.

In treating sprains and strains, procaine injections are used for massaging of hematoma formation, for disruption of adhesions by manipulation, and for restoration of the full range of motion of affected joints.

Low-back complaints are common, congenital anomalies frequently leading to symptoms after strenuous exercise. Persistent complaints are thoroughly investigated.

Attacks of the "bends", often resulting in orthopaedic complications, are guarded against by "washing out" the system with oxygen before each flight, thus lessening the possibility of embolus formation.—*Fred G. Hodgson, M.D., Atlanta, Georgia.*

TRAUMATIC SURGERY. A REVIEW OF SOME BONE AND JOINT INJURIES IN WARTIME. A. G. Ord, Ralph Shackman, and H. L. M. Roualle. *Surgery*, XIV, 651, 1943.

Injuries treated by the authors occurred in service men during training, or, together with some that occurred among women, while in action on the home front. During the years 1940 to 1941, a total of 258 fractures of all types and twenty-one dislocations came under observation of these British surgeons. Standard methods of splinting, with or without skeletal traction, were used.

The authors advocated:

1. Early excision of the radial head where irregularity of the articular surface may lead to a progressive arthritis.
2. Grafting fractures of the scaphoid bone where they have been untreated for six months or longer.
3. Reconstruction of the coraco-acromial joint in severe displacements, rather than simply attempting to restore the acromioclavicular ligaments.
4. Ambulatory treatment in fractures of the lower limbs.
5. Taking repeated roentgenograms, and using a rapid developer, in preference to using the fluoroscope during manipulations or bone-grafting operations.—*Edward L. Compere, M.D., Chicago, Illinois.*

"PULSATING" HEMATOMA (FALSE ANEURYSM). William C. Beck. *War Medicine*, IV, 502, 1943.

This article consists principally of an excellent review of the literature dealing with wounds which may injure an artery without complete severance. The hematoma which forms about this injured artery will "pulsate". Complete transsection of a small artery or vein will result in the formation of a hematoma, but there will be no pulsation because the artery retracts, and the intima rolls up to plug the lumen within a short time after the injury occurs.

"Pulsating" hematoma should be looked for in every puncture wound which is followed by a large swelling. It should be remembered that the pulsation is rarely palpable, but, if the stethoscope is used carefully, the diagnosis can be determined with reasonable certainty. Beck recommends provisional ligation of the tributary artery, followed by a block of the sympathetic nerve supply.—*Edward L. Compere, M.D., Chicago, Illinois.*

Correction

Dr. A. Bruce Gill has called attention to an error which appeared in the footnote to Table I of his article on "Relationship of Legg-Perthes Disease to the Function of the Thyroid Gland", published in the October issue of *The Journal* (Volume XXV, page 897, 1943).

The fourth footnote, "(S) indicates Shim units" should have read, "(S) stands for Shin., and indicates that the method of phosphatase estimation used was that of Shinowara, Reinhart, and Jones".

TABLE OF CONTENTS

(Continued)

PAGE

MARCH FRACTURES.
By *Captain Baxter L. Clement*, Atlantic City, New Jersey 148

DELAYED REDUCTION OF FRACTURES.
By *John R. Moore, M.D.*, Philadelphia, Pennsylvania 151

BRACHIALGIA. A MANIFESTATION OF VARIOUS LESIONS.
By *I. William Nachlas, M.D.*, Baltimore, Maryland 177

A PRACTICAL TECHNIQUE FOR PRESERVING SURGICAL AND ANATOMICAL DISSECTIONS.
By *J. B. deC. M. Saunders, F.R.C.S.*, and *A. H. Rice, M.D.*, San Francisco, California 185

SYMPATHETIC BLOCK IN THE TREATMENT OF LOCAL SHOCK. AN EXPERIMENTAL STUDY.
By *James E. M. Thomson, M.D.*, Lincoln, Nebraska, *Ferdinand Helwig, M.D.*, Kansas City, Missouri, and *Eugene Sire, B.S.*, Lincoln, Nebraska 189

SHOCK IN EXTREMITY SURGERY.
By *Dallas B. Phemister, M.D.*, Chicago, Illinois 197

NEWS NOTES 202

CURRENT LITERATURE 207

ADDRESSES OF CONTRIBUTORS ..... Cover 3

List of Advertisers—January 1944

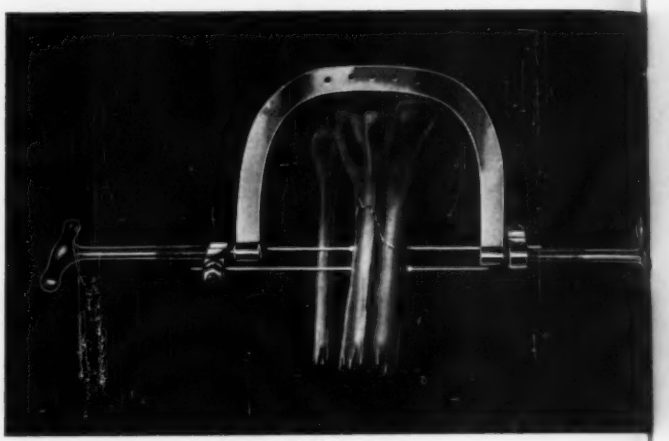
	PAGE		PAGE
Amsterdam Bros., Inc.	6	William H. Kraus	25
Austenal Laboratories, Inc.	12	Lea & Febiger	22
Bard-Parker Company, Inc.	8	J. B. Lippincott Company	23
Bauer & Black	10	Merck & Co., Inc.	7
Julius Berbecker & Sons, Inc.	24	Miller Brace Co.	26
Boston Artificial Limb Co., Inc.	27	The C. V. Mosby Company	28
S. H. Camp and Company	5	Nutrition Research Laboratories	15
Hans W. Christoph, Inc.	24	Price Filler Machine & Mfg. Co.	27
Clay-Adams Co., Inc.	17	The Rumford Press	1
Davis & Geck, Inc.	21	I. Sabel, Inc.	4
DePuy Manufacturing Company	20	J. R. Siebrandt Mfg. Co.	16
Everest & Jennings	27	Singer Sewing Machine Co.	2
E. Fougera & Co., Inc.	13	J. Sklar Manufacturing Company	11
General Electric X-Ray Corporation	3	Thompson Bros. Shoe Co.	4
Hack Shoe Company	14	The Tower Co., Inc.	18
Invalid Walker & Wheel Chair Co.	25	The Max Wocher & Son Co.	26
Johnson & Johnson	9	Zimmer Manufacturing Co.	25
The Kendall Company	10		

De Puy

PEASE BOW with THOMSON BEADED WIRE

Pat. No. 2,024,325 and No. 1,949,969

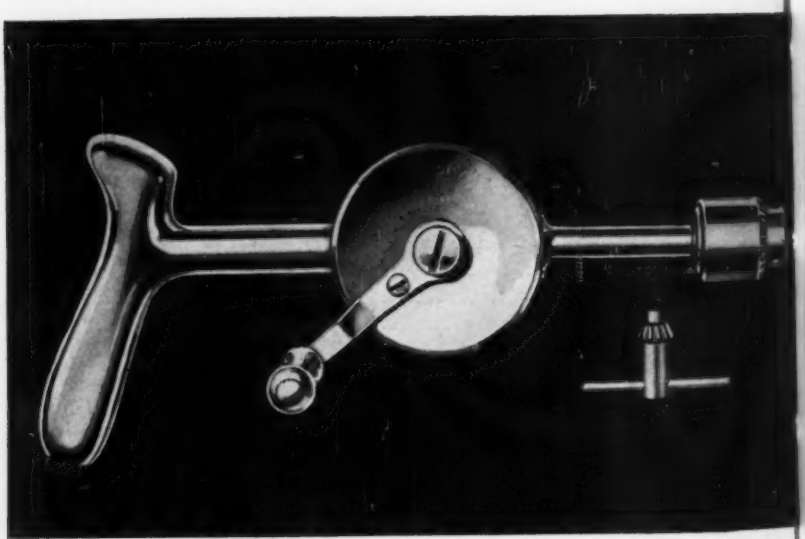
No. 91—Surgeons find that the use of this beaded wire set is simple, fixation adequate, and normal healing processes are favored in certain oblique or special fractures of the lower extremities. The wire is inserted directly through the point of the fracture —will hold fragments from slipping.



PEASE BONE DRILL for BEADED WIRE

No. 384—This drill is cannulated from chuck through the handle to accommodate a beaded wire or Steinmann Pin—any standard size. No guide is needed.

Write for fracture catalog



DEPUY MANUFACTURING COMPANY
WARSAW, INDIANA